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THE HEART SOUNDS.[1]

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DURING the last few years renewed interest has been displayed in the interpretation of the heart sounds. The study of the heart sounds held pride of place as a clinical method, from the time of the introduction of the stethoscope until Mackenzie drew attention to the importance of the cardiac muscle. Prior to his time study was directed towards the description and elucidation of heart murmurs.

Mackenzie rightly stressed the importance of considering the heart muscle in any examination, but after his time there was a tendency to disregard murmurs to some extent. Lately there has again been a swing of the pendulum, and research workers and cardiologists again are intensely interested in heart sounds. This renewed interest has been brought about by the increase of our knowledge of the physiology of the heart and by the introduction of the electrocardiograph and later of the phonocardiograph or the stethograph.

The results of the study of the heart's action, the synchronous recording of the pressures of the heart's chambers and of the heart sounds, and the electrocardiogram have added enormously to the knowledge of the changes taking place during the cardiac cycle.

The Cardiac Cycle.

The succession of changes occurring in the heart during its contraction is referred to as the cardiac cycle. These changes occur very rapidly, so that

[1] Read at a meeting of the Queensland Branch of the British Medical Association on March 3, 1939.

to understand their significance fully special recording apparatus is necessary. Harvey referred to the great difficulty of observing these changes, and it is very interesting to recall his words:

> When I first tried animal experimentation for the purpose of discovering the motions and functions of the heart by actual inspection and not by other people's books, I found it so truly difficult that I almost believed with Frascatorius that the motion of the heart was to be understood by God alone.
>
> I could not really tell when systole or diastole took place, or when or where dilation or constriction took place, because of the quickness of the movement.
>
> In many animals this takes place in the twinkling of an eye, like a flash of lightning. Systole seemed at one here, diastole there, then all reversed, varied and confused.
>
> So I could reach no decision, neither about what I might conclude myself nor believe from others. I did not marvel that Andreas Laurentius wrote that the motion of the heart was as perplexing as the flux and reflux of Euripus was to Aristotle.

These changes in the heart during its action, which so perplexed Harvey, and which all who have watched the heart beating under the X ray screen have found so difficult to evaluate, have been studied very exhaustively in animals and man. Experiments in recording the intraauricular and intraventricular pressures in animals, the recording of aortic and venous pulsation in man by the polygraph, and the results of electrocardiography have enabled scientific workers to build up a not unimposing mass of precise knowledge of the heart's action.

To correlate the sounds that we hear with the stethoscope with what is actually taking place in the heart it is necessary for us to have a mental picture of the cardiac cycle.

We must remember that: (*a*) blood pours into the auricles from the great veins throughout the whole cycle, except for a short period, 0·1 of a second, during which the auricle is actually contracting, that is, the auricular systole; (*b*) filling of the ventricle (in the normal heart with adequate semilunar valves) takes place throughout the cycle, except during actual ventricular contraction and a short period subsequent to this (0·08 of a second), which represents the time between the closing of the semilunar valves (the aortic and pulmonary valves) and the opening of the auriculo-ventricular valves (the mitral and tricuspid valves).

Numerous methods have been used to register the pressure changes of the cardiac chambers and great vessels, including those of Marey, Tick and Hurtle; but the greatest assistance has been rendered by Wigger's modification of Frank's optical method.

Metal capsules, containing a diaphragm and mirror, are inserted into the lumina of the various parts, and changes of pressure are registered by reflected light falling on a moving photographic film. In this way the action of the various chambers of the heart is synchronously recorded. At the same time the electrocardiogram and the phonocardiogram may be registered on the same record.

The Auricular Pressure Curve.

The auricular pressure curve is shown in Figure I. Here there are three positive waves and three negative waves. *A* is due to auricular systole, but registers only a few millimetres of mercury. As the auricle contracts more and more of the fibres

FIGURE I.
Auricular pressure curve. From "Physiological Basis of Medical Practice" (Best and Taylor).

become activated and increase the intraauricular tension; at the summit of the wave the whole auricle is contracting; from then on the fibres relax in a progressive fashion and the pressure falls. The auricular contraction may be divided into a dynamic phase, when the number of contracting fibres is increasing and the tension is rising, and the adynamic phase, when the majority are relaxing and the pressure falls. The duration of each phase is about 0·05 second.

The second wave, *C*, is due to rising pressure in the ventricle. As the ventricle commences to contract the auriculo-ventricular valves are bulged back into the auricles; the summit of the wave corresponds to the opening of the semilunar valves.

The negative wave, *X*, is thought to be due to two factors: (*a*) the lowering of the general intrathoracic pressure by the ejection of the heart's contents, about 60 cubic centimetres of blood, from the thorax; (*b*) the drawing down of the auriculo-ventricular septum as the ventricle contracts.

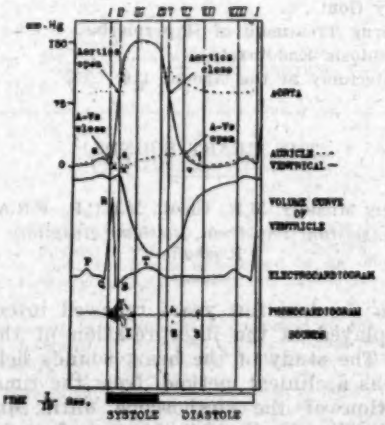

FIGURE II.
The ventricular pressure curve. From "Physiological Basis of Medical Practice" (Best and Taylor).

The third positive wave, *V*, is due to the rise of the pressure in the auricle as the blood pours in from great veins during ventricular systole, that is, while the auriculo-ventricular valves are closed.

The negative wave, *Y*, is due to the fall of the auricular pressure when the auriculo-ventricular valves open. Sometimes a small notch is seen near the summit, which is ascribed to vibrations set up by the closing of the semilunar valves.

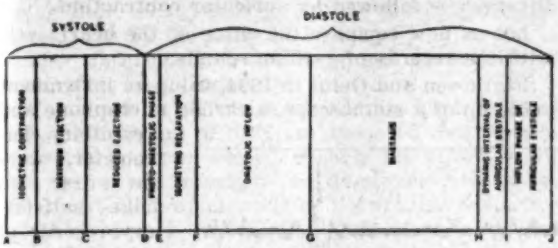

FIGURE III.

Scheme summarizing consecutive phases of the cardiac cycle (after Wiggers). From "Physiological Basis of Medical Practice" (Best and Taylor).

In actual practice the effect of auricular contraction does not appear to account for very much—it is only one-tenth of the cardiac cycle—and when it ceases to contract, as in auricular fibrillation, filling of the ventricle does not appear to be interfered with.

A wave of the auricular curve and is due to auricular systole.

When the ventricle contracts, the pressure of its contents almost immediately rises above the auricular pressure; this causes firm closure of the auriculo-ventricular valves, which actually bulge into the auricular cavity to produce the *C* wave in the auricular pressure curve.

The pressure in the ventricle continues to rise rapidly, giving us the almost perpendicular upstroke of the curve; when it rises to just above the pressure in the aorta and pulmonary artery respectively the semilunar valves are forced open and the blood issues forth from the heart, the ventricular and aortic pressures rising together to the summit of the wave. Then as the ventricles relax there is a rapid fall of pressure. This rapid fall produces an incisura in the aortic curve. As the pressure falls it rapidly reaches a point which is less than the diastolic pressure, and the semilunar valves close. The further relaxation of the ventricle during diastole allows the pressure to fall below the auricular pressure, and the auriculo-ventricular valves open, so that the cavities of the auricle and ventricle freely communicate and allow the blood to pass from the great veins and auricle into the

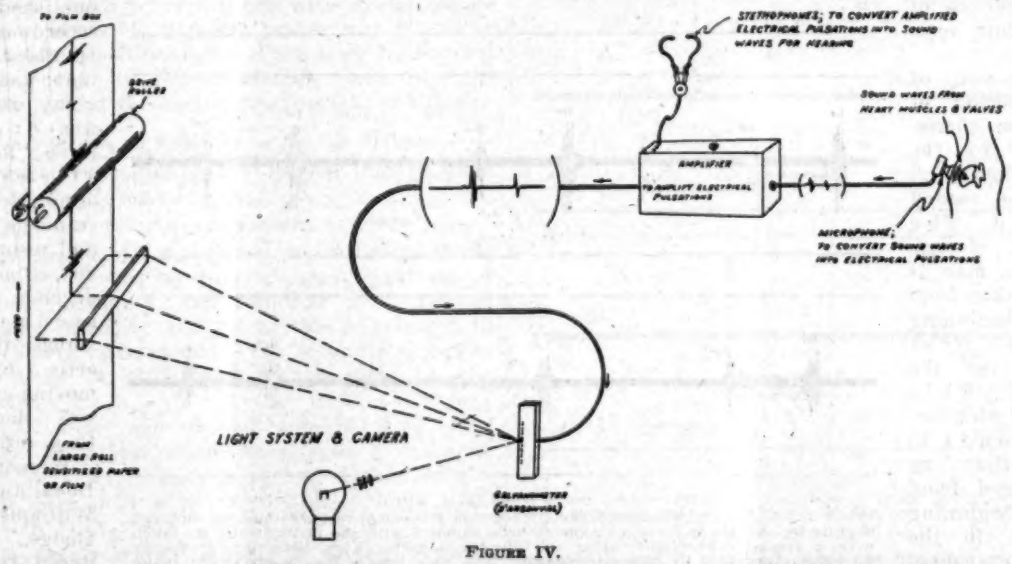

FIGURE IV.

Schematic diagram of the stethograph. The sound waves produced by the patient's heart are picked up by a specially designed microphone, which converts sound pressure waves into electrical pulsations. The pulsations are amplified by a vacuum tube amplifier connected to a galvanometer. Stethophones reconvert the electrical pulsations, amplified many times, back into sound waves to which the ear is sensitive. The electrical pulsations flowing through the galvanometer produce oscillations of the galvanometer coil, upon which a tiny mirror is mounted. Light falling on this mirror is reflected upon moving photographic paper. (Reproduced from M. L. Lockhart's paper in *The American Heart Journal*, July, 1938.)

The Ventricular Pressure Curve.

Knowledge of the ventricular pressure curve is of the greatest assistance in any attempt to understand the why and the wherefore of the heart sounds.

Usually there is a small wave which immediately precedes the ventricular contraction and the main rise of the pressure curve. This corresponds to the ventricle, in which, as it fills, a slight rise in pressure occurs.

From the figure we see that ventricular systole is divided into two periods by the opening of the semilunar valves. The first period is called the presphygmic period or period of isometric contraction; that is, first, a period of ventricular systole which is not registered in the arterial tracing or

blood pressure curve, and, secondly, a period in which the muscular fibres are contracting on an incompressible mass of blood and are not shortened.

The second period of systole is called the sphygmic or ejection period when blood passes from the heart into the aorta and pulmonary artery. The short period at the beginning of diastole following the ejection period and leading to the opening of the auriculo-ventricular valves is called the post-sphygmic period or period of isometric relaxation. Both the semilunar and the auriculo-ventricular valves are closed and no blood is entering the heart, the heart is relaxing, but the fibres are not lengthening.

The first heart sound corresponds to the isometric contraction period and period of maximum ejection.

The end of the period of ejection corresponds to the commencement of the second sound. The period of systole in man is that taken from the beginning of the first sound to the second. This period also corresponds to the time as measured from the beginning of *P* (in the electrocardiogram) to the end of *T*. Wiggers divides the ejection period into a period of maximum ejection and a period of reduced ejection; he refers to that part in the early diastolic period corresponding to the incisura of the carotid pulse and aorta curve just before the opening of the semilunar valves as the protodiastolic period.

After the auriculo-ventricular valves open the pressure rapidly falls. This is the period of "rapid filling" of the heart. This is followed by a period when the ventricle is nearly full, little blood is entering and the pressure is low. This is referred to as the period of diastasis. This again is followed by auricular systole, giving us the first small wave. The period of diastasis lengthens as the heart slows. Diastasis is followed by auricular contraction.

Let us now compare the curve of the heart cycle with the records of cardiac sounds.

Einthoven and Gelut in 1894, using an instrument made up of a stethoscope, a carbon microphone and a capillary electrometer, were the first to make satisfactory records of the heart sounds.

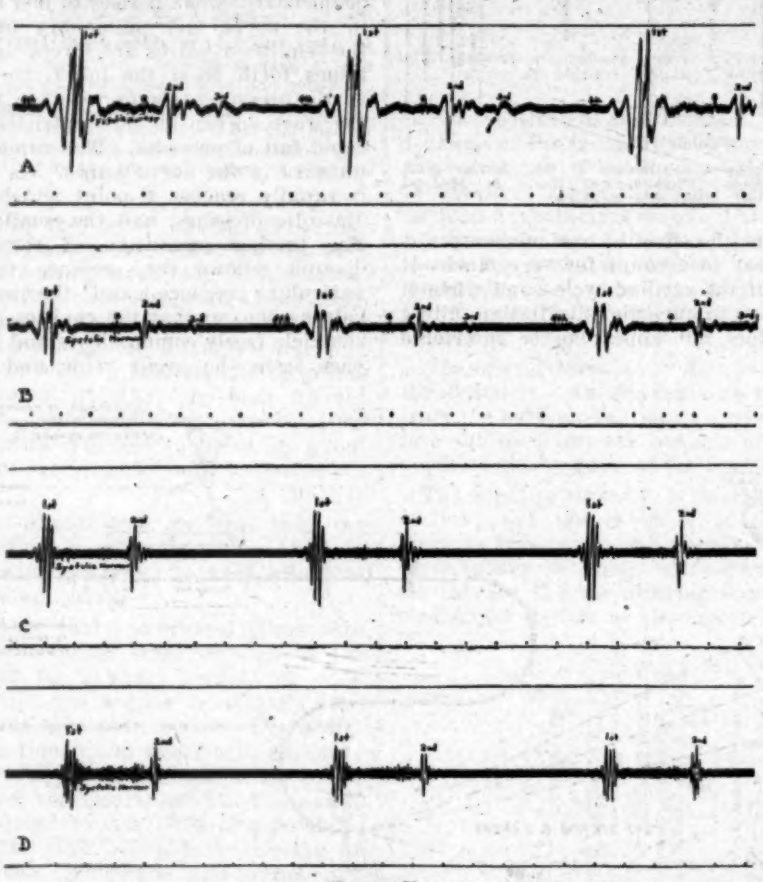

FIGURE V.

Effect of filtering heart sounds. The stethograph frequency response may be modified slightly by the use of three microphone bells supplied with the instrument. *A*: taken with a special microphone, using the number 1 bell. Very little low frequency filtering is provided by this microphone. The base line is too "wavy". *B*: taken with the standard microphone using the number 1 bell. Base line is now straight. *C*: taken with the standard microphone, using the number 2 bell. Some important sounds are filtered out. *D*: taken with the standard microphone, using the number 3 bell. Systolic murmur is magnified. (Reproduced from M. L. Lockhart's paper in *The American Heart Journal*, July, 1938.)

The instrument was later improved by the introduction of the string galvanometer instead of the capillary electrometer. They published their records in 1907. In these early instruments great difficulty was encountered, first in attempts to eliminate extraneous noises, and secondly in an attempt to develop an instrument in which the inertia of the moving parts was decreased to a minimum.

Wiggers and Dean, and later Williams and Dodge, introduced types of sound-recording machines, all of which had certain drawbacks. Lockhart last year introduced his electrostethoscope, which appears to be the most practical instrument of this type so far to come forward (Figure IV).

By the instrument sound waves in the desirable band of frequencies, that is, 75 to 550 cycles per second, are picked up and are amplified. Room noises much above this band are largely excluded.

The human ear has the greatest difficulty in hearing the vibrations at either end of the "sound

spectrum". This explains why the low-pitched diastolic murmur of mitral stenosis must become quite loud before it can be heard. In fact it is not unusual to be able to feel the diastolic thrill before any murmur can be detected, the coarse vibrations being more easily picked up by the sensation of touch than by the sense of hearing.

The vibrations due to the apical thrust of the heart were also eliminated.

Further filtering of the sounds has been brought about by the use of different types of chest pieces.

The stethograph records clearly vibrations below 600 cycles per second. The dominant frequencies of almost all heart sounds and murmurs are below 1,000 cycles per second, and most of them do not exceed 600 cycles per second; that is, the highest pitched sounds heard so well by the ear are only a fraction of the vibrations actually present.

The instrument tends to show up best those low-pitched apical diastolic murmurs which are so difficult to hear under ordinary conditions. Margaret Harper McKee, of New York, examined and made records first of 105 normal children, to establish a basis for comparison, and then examined and recorded the heart sounds of 130 children who had a definite history of rheumatic infection. The records in normal children were taken with a stethoscope bell, two inches in diameter, which was placed in each of three positions: (*a*) at the apex, left lateral position; (*b*) at the pulmonary area, upright position; (*c*) at the aortic area, upright position.

Heart Sounds as Recorded in Normal Children.

These records (Figure VI) showed that in these 105 normal children's hearts:

1. Sinus arrhythmia was present in every case.

2. The first sound was shown to consist of several vibrations preceded by two or three vibrations of increasing intensity, and followed by a few of decreasing intensity and pitch; this is referred to as the fourth heart sound. The second sound was short, lower in pitch, and of the same or less intensity.

3. Split first and second sounds were very common. Split first sounds were found at the apex in 24% of cases, and split second sounds at the base in 35%. In some cases split sounds were present in both areas.

4. The stethogram showed a slight systolic murmur in every case. In only 6% was a systolic murmur audible by ordinary auscultation.

5. In no case at all was a diastolic murmur demonstrated either by auscultation or in the stethographic tracing.

6. Third heart sounds were heard actually in 6% of cases, but were present in every record.

Thayer found 65% of third heart sounds in his studies, and Braun Mendenez 60% in 100 students.

The third heart sound has been the subject of much controversy; the manner of its actual production is not yet settled. Paul White refers to it as being probably due to the opening snap of the mitral valve. Others consider that it is due to the vibrations set up by the blood rushing into the empty ventricle during the period of "rapid filling" which immediately follows the opening of the mitral and tricuspid valves.

The fact that the period between the second sound of the heart and the third sound is always constant in the same reading, and that the mitral diastolic murmur always follows or replaces this sound, rather suggests that it corresponds to the opening of the mitral valve.

This third heart sound is frequently increased in mitral stenosis.

The fourth heart sound is undoubtedly due to auricular activity. Auricular sounds are almost

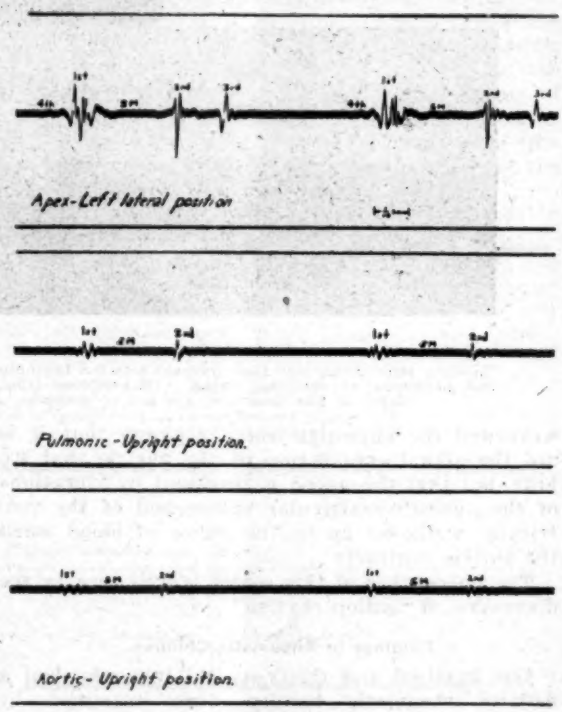

FIGURE VI.
Normal heart sounds. (Reproduced from M. H. McKee's paper in *The American Heart Journal*, July, 1938.)

always heard in cases of complete heart block, when the auricles are beating independently of the ventricles.

Crighton Bramwell has called attention to the part which the auricular sound plays in producing a peculiar roughening of the first sound. This he found to be most noticeable in athletes who were accustomed to long periods of strenuous exercise, such as long-distance swimmers, runners and cyclists. It was not so noticeable in sprinters or middle-distance athletes.

Bramwell has shown by phonogrammatic tracings how the auricular sound accentuates the first sound when the auricular contraction happens to syn-

chronize with the ventricular contraction (Figures VII and VIII).

These tracings also show how the early part of the first sound is made up of these auricular vibrations.

It has been shown that the auricular sound does not bear a fixed relationship to the *P* wave of the electrocardiogram; it usually follows it at some distance, this distance being much greater than that between the *R* wave and the ventricular contraction.

This pause between the *P* wave and the auricular sound suggests that it is not the actual contraction of the auricle that we hear, but that the sound is produced by vibrations of the auriculo-ventricular valves and of the ventricular walls set up by the inflow of blood when the auricle contracts.

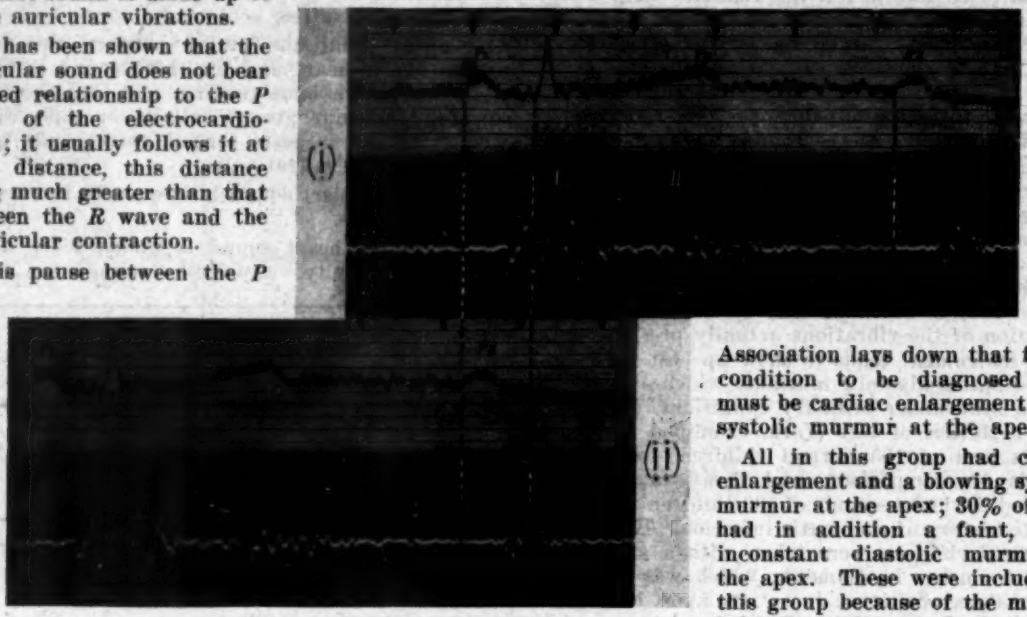

FIGURE VII.
Showing relationship and time interval between beginning of first heart sound and beginning of auricular sound. (Reproduced from Crighton Bramwell's paper in *The Quarterly Journal of Medicine*, April, 1935.)

The importance of this sound is well seen in the discussion of "gallop rhythm".

Findings in Rheumatic Children.

One hundred and thirty-six children who had a definite rheumatic history were examined by Margaret McKee. All the children had had polyarthritis, chorea, or a carditis of a rheumatic type. The cases were divided into groups according to the classification of the New York Association: (*a*) cases of possible or potential heart disease, (*b*) cases of mitral incompetence, (*c*) cases of mitral incompetence and mitral stenosis, (*d*) cases of mitral stenosis and aortic incompetence, (*e*) cases of mitral stenosis and aortic stenosis.

Potential Heart Disease.—There were eleven patients in this group of possible and potential rheumatic heart disease according to the classification of the New York Association. All had had rheumatism; all had systolic murmurs. There was no cardiac enlargement, either clinically or radiographically. The systolic murmurs in the tracings were exactly the same as those which were audible in normal children (Figure IX). One case showed only some waving of the base line after the second sound, but no diastolic murmur was ever heard.

Mitral Incompetence.—The New York Heart Association lays down that for the condition to be diagnosed there must be cardiac enlargement and a systolic murmur at the apex.

All in this group had cardiac enlargement and a blowing systolic murmur at the apex; 30% of them had in addition a faint, short, inconstant diastolic murmur at the apex. These were included in this group because of the murmur being inconstant and of the fact that there was no evidence of an enlarged left auricle. In five cases the second pulmonary sound was increased in intensity, that is, accentuated pulmonary second sound of ordinary auscultation.

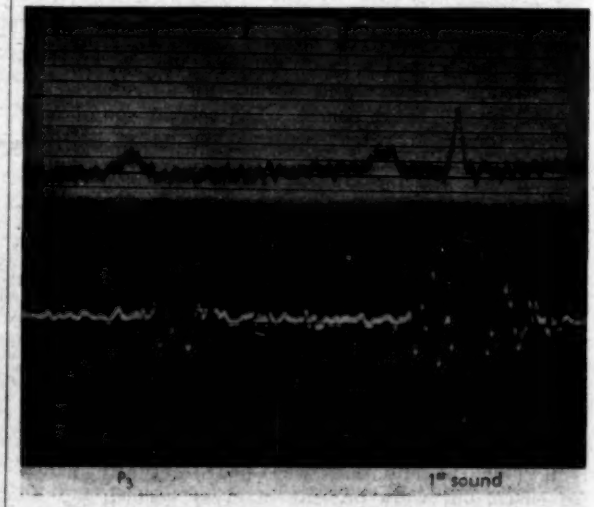

FIGURE VIII.
(Reproduced from Crighton Bramwell's paper in *The Quarterly Journal of Medicine*, April, 1935.)

The systolic murmurs were most intense early in systole and tended to fade out. Diastolic murmurs occurred in 86%. These diastolic murmurs were

faint. They followed the third heart sound, their sounds changed with respiration, sometimes there was a third heart sound, and sometimes a diastolic murmur.

Mitral Insufficiency and Questionable Mitral Stenosis.—There were six in the group of mitral insufficiency and questionable mitral stenosis, which

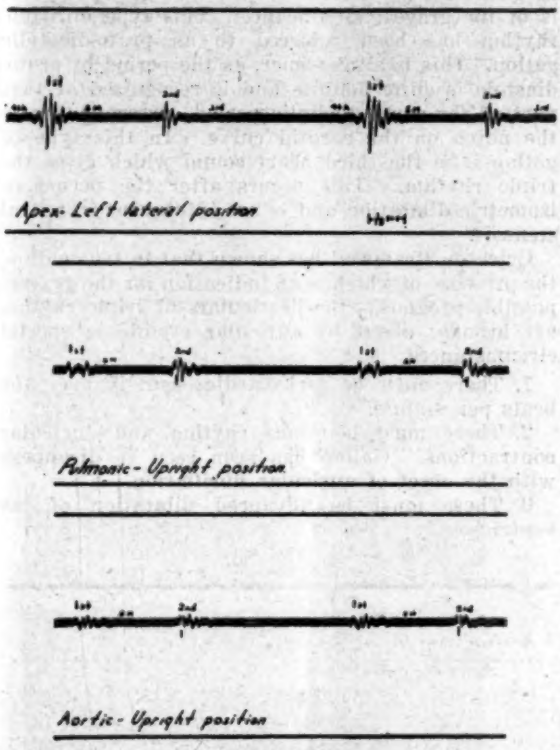

FIGURE IX.

Heart sounds of a patient with potential and possible heart disease, having a history of rheumatic fever, and clinically showing a systolic murmur as the only possible evidence of cardiac damage. The record is like that of a normal heart. (Reproduced from M. H. McKee's paper in *The American Heart Journal*, July, 1938.)

were divided into: (*a*) those having a systolic and a short diastolic murmur at the apex—the diastolic murmur was not of a rumbling character, but there were signs of cardiac enlargement; (*b*) those having an apical systolic murmur and a rumbling diastolic murmur, but no evidence of cardiac enlargement.

Mitral Insufficiency and Stenosis.—The group of mitral insufficiency and stenosis could include many of the previous group, but it was reserved for those cases, 57 in number, in which there were a loud systolic and a diastolic murmur at the apex, with definite signs of cardiac enlargement. Of these, 14% showed definite accentuation of the pulmonary second sound.

The systolic murmurs were all loud, some being louder than the first sound. The diastolic murmurs varied somewhat (Figure XI). These diastolic murmurs were louder than in the previous group, and extended right through diastole from the third heart sound to the fourth heart sound. In three cases there was an increase of the intensity of the diastolic murmur in the presystolic period. In 12% of cases there was evidence of a sound, as shown by large vibrations between the auricular sound and the first sound. This corresponds to the presystolic murmur so assiduously searched for by the careful examiner. These records were taken with the patient at rest; it is suggested that if the patients had been exercised this presystolic accentuation would have shown out more frequently.

From these records we can see that the characteristic murmur of mitral stenosis is a diastolic murmur, which commences at the time of the third heart sound, continues through diastole and may or may not have the crescendo presystolic ending.

There is always a definite space between the second sound and the beginning of the diastolic murmur. This silent period corresponds to the isometric relaxation period between the closing of the semilunar valves and the auriculo-ventricular valves; in others words, the time between the closing of the aortic valves and the opening of the

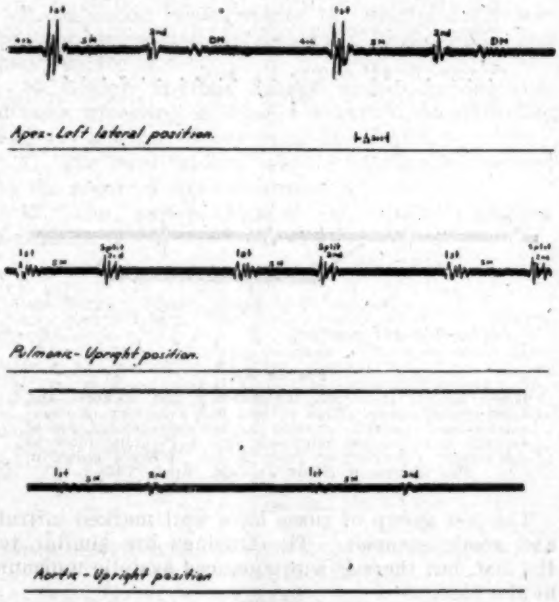

FIGURE X.

Heart sounds in mitral insufficiency. This patient gave a history of rheumatic fever and clinically showed cardiac enlargement, apical systolic murmur, and an inconstant early faint apical diastolic murmur. (Reproduced from M. H. McKee's paper in *The American Heart Journal*, July, 1938.)

mitral valves. This pause helps sometimes to distinguish a mitral from an aortic diastolic murmur.

Mitral Stenosis and Aortic Deficiency.—The next group are those with mitral stenosis and aortic deficiency. Sixteen patients were in this group. Here clinically, in addition to the systolic and diastolic murmurs at the apex, there was present a high-pitched blow in the aortic area during

diastole, following immediately the second sound (Figure XI).

The stethograph tracing in these cases shows the diastolic murmur badly because of its low intensity. The low intensity of this murmur makes it difficult to record, though it can be heard quite well with the stethoscope.

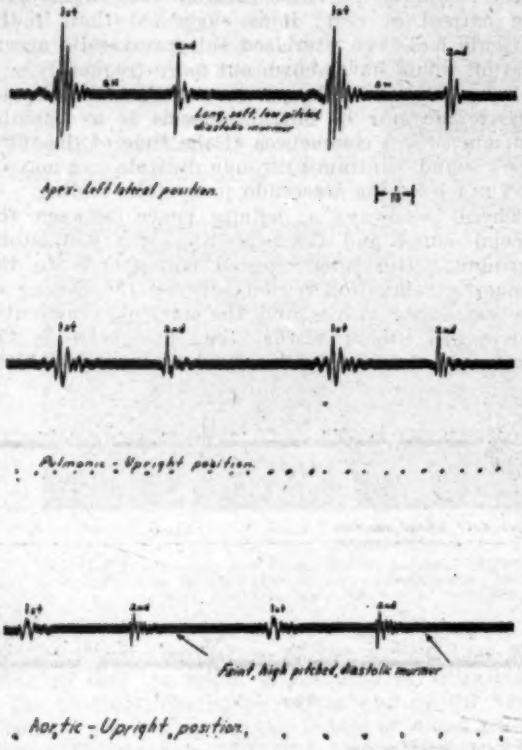

FIGURE XI.

Heart sounds in mitral insufficiency and stenosis and aortic insufficiency. This patient had a history of rheumatism and clinically showed cardiac enlargement, apical systolic and diastolic murmurs, and a diastolic blow at the base. (Reproduced from M. H. McKee's paper in *The American Heart Journal*, April, 1938.)

The last group of cases have well marked mitral and aortic stenosis. The tracings are similar to the last, but there is a pronounced systolic murmur at the base.

Tracings were also taken in certain cases during the attacks of rheumatic carditis. The sinus arrhythmia in 30% disappeared from time to time, and in the others was less marked. This is partly but not wholly obtained by the increase in rate which usually accompanies carditis. The tracings show that the increased rate occurs at the expense of the diastolic period, chiefly of the period of diastasis.

During active rheumatic carditis there is an increase in any murmurs present, and the murmurs may change. They usually become louder and more highly pitched. In some cases the stethoscope suggested the presence of gallop rhythm, that is, there was a definite triple rhythm (Figure XII). This is shown in such a case to be due to an accentuation of the normal third heart sound, with some weakening of the first heart sound. This should not really be referred to as gallop. It is much better to give this the term "triple rhythm" and to reserve the term gallop for those grave cases in which the production of this true gallop rhythm is of the gravest significance. This type of triple rhythm has been referred to as proto-diastolic gallop. This is a misnomer, as the period of proto-diastole is quite definite and is recognized at that part of the early diastolic period corresponding to the notch on the carotid curve. In this type of gallop it is the third heart sound which gives the triple rhythm. This occurs after the period of isometric dilatation and is not infrequent in mitral stenosis.

Crichton Bramwell has shown that in true gallop, the presence of which is an indication for the gravest possible prognosis, the particulars of triple rhythm are brought about by auricular systole in special circumstances.

1. There must be tachycardia, usually over 100 beats per minute.
2. There must be sinus rhythm and auricular contractions. Gallop has been seen to disappear with the onset of auricular fibrillation.
3. There must be advanced dilatation of the ventricles.

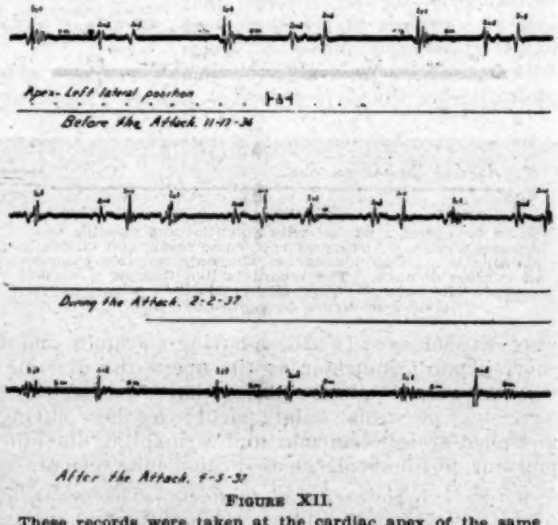

FIGURE XII.

These records were taken at the cardiac apex of the same patient. The first was taken before, the second during, and the third following an attack of rheumatic fever. At the time of the second record the patient was considered to have gallop rhythm. (Reproduced from M. H. McKee's paper in *The American Heart Journal*, July, 1938.)

4. Mitral stenosis is not present. This type of gallop makes up a definite clinical entity, and we should reserve the term "gallop" for it. The term "gallop" should not be loosely used and should rank with the term "angina". This again should be reserved for a definite clinical entity and should never be used except to express the pain from

coronary incompetence, and should thus bear with it its grave prognosis.

In true "gallop" the third sound is probably due to a marked increase of the ordinary fourth heart sound or auricular sound, it is usually associated with a lengthening of the *P-R* interval, so that the auricular contraction approaches the time of

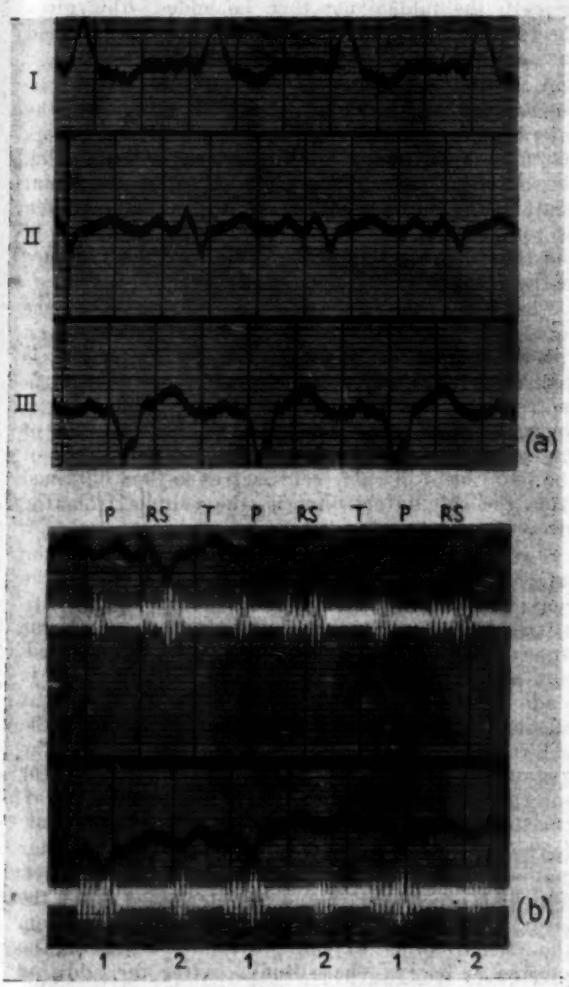

FIGURE XIII.

Diagram showing gallop rhythm. The *P-R* interval is lengthened and the auricular sound is seen to precede the first heart sound. (Reproduced from Crighton Bramwell's paper in *The Quarterly Journal of Medicine*, July, 1938.)

opening of the auriculo-ventricular valves; that is, the auricular contraction takes place during the period of "rapid filling" and the flow of the blood is rapidly ejected into the dilated ventricle.

The vibrations set up under these conditions produce this form of triple rhythm.

Summary.

1. During the last few years there has been renewed interest in the study of heart sounds and murmurs.

2. Much light has been thrown on the actual happenings of the cardiac cycle by the synchronous recording of the intraauricular and intraventricular pressures, the electrocardiogram and the phonocardiogram.

3. Lockhart's electrostethograph has been shown to be of the greatest help in the study of the heart sounds.

4. Margaret McKee has shown that sinus arrhythmia and systolic murmurs are normal in the hearts of healthy children.

5. The third and fourth heart sounds are present in all sound tracings of normal hearts.

6. Diastolic murmurs never occur in healthy hearts under ordinary conditions.

7. Mitral diastolic murmurs in mitral stenosis always follow immediately or replace the normal third heart sound.

8. In mitral stenosis there is a period of relative silence between the second heart sound and the diastolic murmur corresponding to the time in the cardiac cycle between the closing of the semilunar valves and the opening of the auriculo-ventricular valves.

9. In aortic incompetence the diastolic murmur follows the second aortic sound immediately and may replace it.

10. Gallop rhythm occurs under certain conditions involving a dilated ventricle, tachycardia, and the absence of auricular fibrillation.

11. The term "gallop" should be strictly reserved to the above clinical syndrome.

12. True gallop rhythm is of the gravest significance.

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SOME OBSERVATIONS UPON PELVICEPHALOMETRY IN LATE PREGNANCY.

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THE following is a résumé of fifty pelvicephalometric measurements carried out at the Women's Hospital, Crown Street, Sydney, and a brief summary of the conclusions resulting from this method of investigation during the later stages of pregnancy.

The radiographic examination of the pregnant woman is of great value to the obstetrician as an aid to the diagnosis of position, presentation and

lie of the fœtus, as to whether the pregnancy is a single or a multiple one, or as to whether some skeletal abnormalities of the mother or fœtus may be present.

The minor degrees of pelvic contraction or cephalic enlargement cannot be estimated by simple radiography, for it is obvious that an X ray film is a shadowgraph and must follow the enlargement distortion rule of all shadows. For this reason, plain radiography is a very poor index indeed of disproportion, unless there is pronounced pelvic deformity or hydrocephalus.

The fifty patients under review were examined because there was a reasonable suspicion, founded upon the clinical history or clinical findings, to suggest the presence of disproportion or deformity of the maternal pelvis.

Many methods of X ray pelvimetry have been devised, but the method of investigation chosen in these cases appeared to offer a greater amount of information than would be obtained by pelvimetry alone, especially as the fœtal head was directly compared with the pelvic dimensions through which it had to pass during labour.

It is a method which is based upon the volume of the fœtal head compared with the volume capacity of the pelvic diameters, and was first worked out by Robert P. Ball and S. Marchbanks, and published in *Radiology* of January, 1935. The film magnification of the shadows is corrected by a special calculator, the Ball pelvicephalometer. Briefly the method is as follows.

Two films are taken under the standard conditions of a thirty-inch distance between focal spot and film, as set down in the technique of Ball and Marchbanks. One is a true antero-posterior film and the other a true lateral film. Both films include all the bony landmarks of the maternal pelvis, and should clearly define the whole of the fœtal skull. In breech presentations additional films must be taken.

The usual observations regarding abnormalities, presentation of the fœtus, degree of engagement and flexion of the head, position of the limbs *et cetera* are first noted. Base lines are then drawn upon each film. In the antero-posterior film the base line passes vertically upwards through the mid-point of the greater trochanter of the femur, and in the lateral film the base line is drawn vertically, tangential to the most posterior bony point of the sacrum. It is to these base lines that all the pelvic measurements are made, and they represent a fixed plane in reference to the top of the X ray table upon which the patient is lying in the lateral and antero-posterior views. It has been found experimentally that the position of the base lines corresponds with the plane of the table top, if allowance is made for the lateral enlargement distortion of the shadows superficial to them upon the two films.

The circumference of the fœtal skull, as shown in the antero-posterior film, is measured in centimetres by a tracing of the perimeter of the image. The distance in centimetres from the central point of the fœtal head to the table top in the antero-posterior direction is determined by measurement on the lateral film of the distance from the central point of the fœtal skull to the spine of the sacrum (base line).

The distance in centimetres from the table top to the film lying on the Bucky diaphragm tray beneath the table must then be added; this gives the total distance from the centre of the fœtal head to the film in the antero-posterior view.

A similar procedure is carried out on the circumference of the fœtal skull, as shown in the lateral view; the distance of its central point from the film is measured on the antero-posterior film, the distance from the central point of the fœtal skull to the mid-trochanteric base line being taken as representing its distance from the table top in the lateral projection. The table top film distance is then added.

The two measurements of the perimeter of the fœtal skull represent the circumference of a spheroid, from which the mean circumference is determined. To the mean circumference two centimetres are added, for it has been found experimentally to be a reliable index of the thickness of the soft tissues of the fœtal scalp. The volume of the fœtal head in cubic centimetres is then obtained from the calculator—that is, the volume from the circumference.

Two pelvic measurements are then made, the internal conjugate and the biischial diameters. The conjugate on the lateral film is measured in centimetres from the sacral promontory to the posterior superior border of the *symphysis pubis*. The estimation of the object-film distance of this diameter is made on the antero-posterior film by measurement of the distance from the *symphysis pubis* to the mid-trochanteric base line and addition of the distance from table top to film. The correction for enlargement distortion is then made on the calculator and the true diameter (conjugate) is read.

The biischial diameter is measured in a similar manner on the antero-posterior film, and the object-film distance of this plane is determined from the lateral film by measurement of the distance from the ischial spine to the posterior border of the sacrum or coccyx (base line). After the addition to this measurement of the distance from table top to film, the linear magnification of this diameter is reduced by the calculator to its true length.

The volume capacity of the least pelvic diameter, which may be either the inlet or outlet, can then be translated into a sphere by a direct reading from the calculator—that is, the volume from the diameter.

It has been stated by Ball that the pelvic measurements are accurate to within five millimetres and that the mean circumference of the fœtal skull is accurate to within one centimetre in the majority and to within two centimetres in all cases.

Important points in the technique are that the bladder and rectum should be empty at the time the

films are taken, that a soft abdominal binder should be applied, and that the estimated date of labour should be known to the radiologist.

Results and Commentary.

In this series of fifty cases twelve Cæsarean sections were found to be necessary. In eight of them the X ray report confirmed the clinical findings of disproportion. In one case of the remaining four the fœtus was found to be in a left occipito-posterior position, which persisted. The measurements in this case indicated that there was ample room, had rotation into an occipito-anterior position been possible. In a second case, in which the measurements indicated that labour should have progressed normally, the clinical notes stated that "trial labour was ineffective, the pains were very slight and the uterus tense".

The remaining two cases suggest the failure of the method, since the measurements indicated that the fœtal head should pass the least pelvic dimension; yet in one case the external biischial estimation would barely accommodate three fingers. I can offer no satisfactory explanation of these cases, except to suggest a technical error, since they were among the earlier examinations carried out in this series. A further possible explanation will be suggested later.

No case in this series was encountered in which a definite disproportion was indicated radiologically but a normal or forceps delivery was effected.

In three cases in which the difference in the volume of the fœtal skull to the volume capacity of the least pelvic dimension was approximately 100 cubic centimetres, there was a reasonable prediction of a difficult labour. In all three cases delivery was normal.

It is worthy of note at this stage that in the limited literature available upon the degree of safe moulding of the fœtal head a tentative limit of 150 cubic centimetres has been set down by the American observers. I am inclined to be conservative in this regard, and one of the following cases will support this view.

On the other side of the ledger four cases must appear. Two of these patients had had a previous Cæsarean section, but the pelvicephalometric measurements indicated that there was no apparent reason why a normal labour should not occur. Trial labour in both these cases resulted in a normal birth, one being a forceps delivery. In the third case there was already a volume difference of 140 cubic centimetres between the fœtal skull and the volume capacity of the pelvic outlet; these facts, in my opinion, definitely indicated the necessity for excessive moulding and compression. The child died four days after a difficult forceps delivery, presumably from cerebral injury, but a *post mortem* examination was not obtainable.

In the fourth case measurements were not made before labour, but the maternal pelvic measurements were carried out after forceps delivery of a still-born fœtus. It was then found that the biischial diameter of the pelvis was nine centimetres, the volume capacity being only 380 cubic centimetres—the same measurements as in the previous case.

In the remaining thirty-one cases the X ray examination was carried out because there was a suspicion of clinical disproportion, a history of a previous difficult labour, or suspected maternal pelvic or fœtal abnormality. Eight of the births were recorded as being forceps deliveries and the remaining twenty-three were normal. The X ray report in each case indicated ample room for normal delivery.

Several of these cases are interesting in regard to the conditions demonstrated. Two were of breech presentations and one of face presentation; after correction into occipito-anterior positions normal births resulted. In a fourth case there was a congenital dislocation of the left hip joint of the mother; but clinically and radiologically there was no evidence of disproportion. The proportion of contracted inlet to contracted outlet in this series was as one is to five. Eight patients had an equal conjugate and biischial diameter.

From the observations in this series there could be no exact prediction as to the birth weight of the child, for in some cases labour did not occur for two to four weeks after the measurements had been taken; but there is no doubt that an approximate weight of the fœtus can be given from the dimensions of its skull. It has been noted, for instance, that if the fœtal skull has an estimated volume of approximately 540 cubic centimetres within a few days of labour, the birth weight of the infant will be between seven and a half and eight and a half pounds, according to its nutrition.

The type of pelvis according to the classification of Caldwell and Moloy cannot be given with any exactitude with this particular technique, because the central ray does not pass perpendicularly to the plane of the inlet in the antero-posterior view, and the reduction of the shadow enlargement distortion of the transverse diameter of the inlet can be only approximate, since there is no fixed bony point corresponding to it from which measurements can be made in the standard lateral projection. The angle of the sacro-sciatic notch and the curvature of the sacrum can be gauged and the internal conjugate can be measured; but special views for the sub-pubic angle and the plane of the brim are necessary to conform to the standard set down by these authorities.

It may be stated here that a recent publication by R. P. Ball, in *Radiology* of August, 1938, simplifies some of the problems encountered in this series, in which his original technique was applied.

The Ball pelvicephalometric method deals primarily with the conjugate and biischial diameters, and the volume of a sphere having a similar diameter; but in certain types of pelves other diameters are more important than the conjugate in estimating the true volume capacity of

the inlet. For example, the transverse diameter is more important in the anthropoid type of pelvis, and an erroneous calculation of its volume capacity may be made if based upon a measurement of the conjugate diameter alone. In addition, the prominence of the lumbo-sacral junction, the flatness or the accentuated curvature of the sacrum, the relative position and mobility or fusion of the sacro-coccygeal joint, the prominence of the ischial spines, the laxity or sclerosis of the sacro-iliac synchondroses, and even the mobility of the *symphysis pubis*, all play their part irrespective of the actual measurements of the conjugate of the inlet and the biischial diameter of the outlet. I believe that the above facts must be taken into consideration and may form part of the explanation in regard to the two cases under review, in which Cæsarean section had to be performed when there appeared to be a reasonable prediction of a natural birth.

I consider that additional films of the plane of the pelvic brim and sub-pubic angle are necessary, as in this way the type of inlet and asymmetrical variations can be demonstrated, as well as the outlet and the relative prominence of the ischial spines.

Many factors play their part in the mechanism of labour, and it is obviously impossible to devise a method which will fulfil all conditions.

As I have indicated, there have been two apparent failures in the above series; yet even if the margin of error should prove to be greater than 4% when the results of a larger series of cases are completed and published, I think that the method, with certain additional modifications in technique, can be used with advantage as an aid to the subsequent management in the doubtful obstetrical case.

An important feature of this method is the determination of the volume increase in the fœtal skull during the later period of gestation, and this can be ascertained only by repetition of the examination at weekly intervals. A sufficient number of such reexaminations has not yet been completed to allow any conclusions or definite figures to be included in this résumé; but the importance of this can be judged by one of the cases in this series, in which a volume increase of 120 cubic centimetres occurred in fourteen days, resulting in a definite disproportion and Cæsarean section.

The rate of increase may therefore, in some cases, be the determining factor between a difficult labour and a definite disproportion, and in my opinion the measurements should be carried out as close to term as possible, or at a stage when, clinically, induction of labour would be contemplated.

The maximum degree of moulding of the fœtal head which is possible without mortality would naturally depend on many factors, and it is obviously unlikely that any constant figure could possibly be obtained. For practical purposes moulding up to 100 cubic centimetres can safely occur; that is to say that a fœtal skull with a volume greater than the volume capacity of the least pelvic dimension by 100 cubic centimetres will pass normally, but volumes greater than 100 cubic centimetres and up to 150 cubic centimetres should be regarded with increasing suspicion and the clinical course closely observed.

If the significance of the above remarks is realized it will be evident that the radiologist must be reserved in his judgement of disproportion, for it is conceivable that moulding to the extent of 140 cubic centimetres would in certain circumstances be without damage, yet in another case 120 cubic centimetres' difference might end in over-compression. In addition, there are always the margin of error, as stated by Ball himself, and the time that elapses between the measurements and the onset of labour. This I consider to be a most important point.

If the obstetrician seeks some infallible method or methods of measurement he will doubtless never find them, for even with varied radiological aid many of the factors must still remain unseen. In many cases there can be no accurate prediction of the interaction of the powers, passages and passenger, and it is quite obvious that pelvimetry and fœtal cephalometry, if considered alone, can be as misleading as the older method of external pelvimetry unless they be considered as a link in the chain of exact clinical observations. In this regard it has been the practice at the Women's Hospital, Crown Street, Sydney, where all facilities are available, to consider induction or to allow trial labour in all borderline cases and follow, if necessary, with Cæsarean section.

SOME STATISTICAL FACTS CONCERNING WHOOPING COUGH IN NEW SOUTH WALES.

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IF we are to judge by the mortality caused by whooping cough and measles, the traditional fear of diphtheria and scarlet fever is now no more justifiable than the indifference shown by public opinion with regard to the two former diseases. The general public still displays an attitude of indifference to whooping cough which contrasts strangely with the fact that it is undoubtedly one of the most distressing and fatal of the acute infections of childhood. Even if the mortality rate is not now as high as it was in former times, still the number of deaths occurring every year is alarming.

In the period from 1889 to 1938 there have been 27,108 deaths in New South Wales from the four common infectious diseases of childhood (diphtheria, scarlet fever, whooping cough and measles), of which 20,940 (77·3%) occurred among children

under the age of five years. Of the total deaths, whooping cough has been responsible for 8,727, a number which is exceeded only by the number of deaths due to diphtheria (11,849). In the "under five years" age group whooping cough has been the cause of 8,438 deaths, which exceeds the 8,025 deaths due to diphtheria in the same age group.

TABLE I.

Deaths from Diphtheria, Scarlet Fever, Whooping Cough and Measles in New South Wales since 1889, showing Deaths of Children aged under Five Years and Total Deaths.

Year.	Diphtheria.		Scarlet Fever.		Whooping Cough.		Measles.		Total for All Ages.
	Under Five Years.	Total All Ages.	Under Five Years.	Total All Ages.	Under Five Years.	Total All Ages.	Under Five Years.	Total All Ages.	
1889 to 1899	2,902	4,353	546	826	2,413	2,493	1,272	1,625	9,297
1900 to 1909	907	1,400	193	384	1,582	1,643	299	369	3,796
1910 to 1919	1,673	2,535	169	358	1,605	1,646	812	1,095	5,634
1920 to 1929	1,542	1,983	217	460	1,798	1,868	587	786	5,097
1930 to 1938	1,001	1,578	144	294	1,040	1,077	238	335	3,284
Totals	8,025	11,849	1,269	2,322	8,438	8,727	3,208	4,210	27,108

Whooping cough, like measles, rarely causes death, except through its complications, particularly bronchopneumonia. Although the Bertillon rules, according to which a death caused by a complication is attributed to the original disease, are applicable in New South Wales, there must be many deaths due to bronchopneumonia as a complication of whooping cough in which the original disease has never been diagnosed. Therefore, the number of deaths reported as due to whooping cough would be minimal only.

Figure I reveals graphically that the four infections of childhood show a definite trend towards smaller death rates. This is in keeping with the experiences of other countries. At the same time Table II shows that whooping cough has

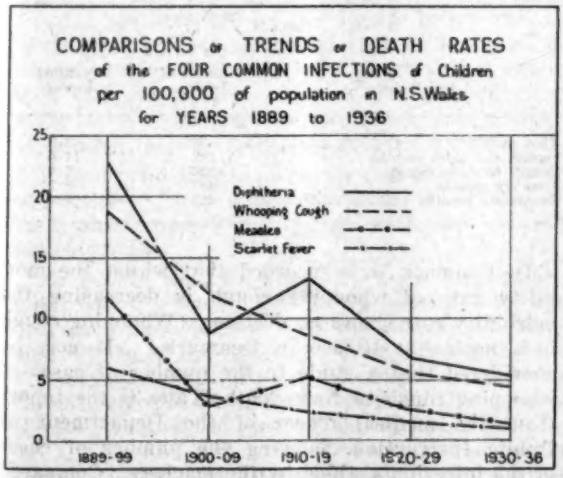

FIGURE I.

increased its proportion of deaths compared with the other infections. It can be seen that whereas for the period from 1889 to 1919 whooping cough was responsible for 33·1% of the deaths, for the period from 1920 to 1936 the percentage had increased to 35·2. Deaths due to scarlet fever had also slightly increased. The proportion of deaths from diphtheria decreased slightly, as also did the proportion of those due to measles.

Although the differences in the percentages for the two groups of years are not very great, still it is only a matter of time and public response to immunization before the deaths due to diphtheria are greatly reduced (as has been actually shown in other countries) and whooping cough takes the lead with the greatest death rate.

TABLE II.

Deaths from Diphtheria, Scarlet Fever, Whooping Cough and Measles, expressed as Percentages of Total Number, due to these Four Infections.

Years.	Diphtheria. (Percentage.)	Scarlet Fever. (Percentage.)	Whooping Cough. (Percentage.)	Measles. (Percentage.)
1889 to 1919 ..	42·8	8·4	33·1	15·7
1920 to 1936 ..	41·4	9·2	35·2	14·2

It is interesting to note that whooping cough during the intervals between the periods from 1901 to 1905 and from 1926 to 1930 had increased its

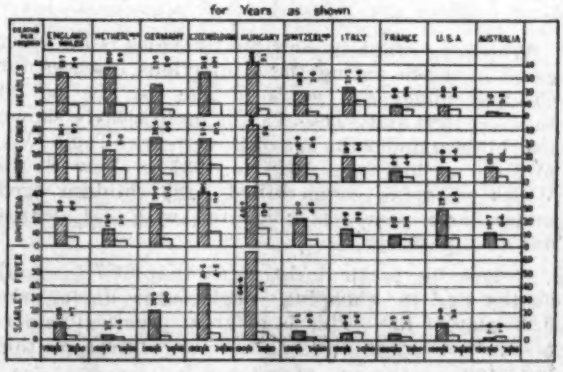

FIGURE II.

relative importance as a factor in the causes of deaths from infectious diseases in six countries, and decreased its relative importance in four (Figure II). Between 1901 and 1905 the position

occupied by whooping cough as a cause of death was first in one country, second in six, third in two and fourth in one only. Between 1926 and 1930 its relative importance had increased, since it occupied first place in four countries, second place in five and third place in one. In six countries it was responsible for a third or more of the deaths from the four diseases. In 1935 it was reported that whooping cough had caused 4,290 deaths in the United States of America, compared with 3,620 deaths from diphtheria. This makes the fifth country in which whooping cough has taken the lead.

Table III shows the number of deaths each year from 1920 to 1938 for the four infections. For this period diphtheria was the cause of 3,561 deaths, in comparison with those due to whooping cough, which numbered 2,945 deaths. In 1920 whooping cough was the cause of 369 deaths, a number which was not attained by any of the other infectious diseases in any one year during this period.

TABLE III.

Deaths from Diphtheria, Scarlet Fever, Whooping Cough and Measles in New South Wales for the years 1920 to 1938 (all ages).

Year.	Diphtheria.	Scarlet Fever.	Whooping Cough.	Measles.
1920 ..	264	24	369	189
1921 ..	306	8	257	39
1922 ..	211	10	92	16
1923 ..	176	13	86	138
1924 ..	225	20	83	36
1925 ..	114	27	323	30
1926 ..	169	115	211	20
1927 ..	143	53	132	90
1928 ..	168	105	103	162
1929 ..	207	78	212	66
1930 ..	175	54	164	100
1931 ..	168	36	186	29
1932 ..	160	57	61	14
1933 ..	104	55	25	45
1934 ..	106	19	286	34
1935 ..	109	18	63	83
1936 ..	133	26	120	22
1937 ..	143	17	93	8
1938 ..	156	12	79	—
Total ..	3,561	754	2,945	1,121

Whooping cough in New South Wales is the chief cause of death among these infectious diseases for children under five years of age for the period between 1889 and 1938. This is more clearly shown for children under the age of three years (Table IV). For the period from 1928 to 1937 in New South Wales whooping cough killed 1,189 children aged under three years, as compared with 754 deaths due to diphtheria.

It is to be particularly noted that 61·7% of all deaths due to whooping cough occur during the first year of life. This is of great practical importance in view of any scheme for control of this infection.

Table IV shows age distribution of deaths from whooping cough and diphtheria from 1928 to 1937 in New South Wales.

According to Madsen,[1] in Denmark in the years from 1910 to 1919 the following deaths occurred

TABLE IV.

Age Period.	Whooping Cough.		Diphtheria.	
	Deaths.	Percentage.	Deaths.	Percentage.
First six months	487	37·1	27	1·5
Second six months ..	323	24·6	118	6·6
Total first year ..	810	61·7	145	8·1
Second and third years ..	379	28·8	609	33·9
Total under 3 years..	1,189	90·5	754	42·0
Total under 5 years..	1,272	97·9	1,206	67·1
Total under 15 years..	1,301	99·0	1,710	95·1
Total for all ages ..	1,313	100·0	1,797	100·0

from whooping cough per 100,000 children born alive: eight died during the first month (*circa* eight per month); 75 died during the second and third months (*circa* 37 per month); 424 died during the period from the third to the twelfth month (*circa* 47 per month).

Madsen comes to the conclusion that these figures "prove the old saying about the relative immunity, whether congenital or due to other causes, of the first month and the dangers of the second half of the first year".

An analysis of 810 deaths in the first year of life due to whooping cough in New South Wales during the years from 1928 to 1937 is interesting, inasmuch as the results obtained do not support Madsen's conclusions (see Table V).

TABLE V.

Age Period.	Number of Deaths.	Monthly Average.
First month	28	2·8
Second and third months	232	11·6
Fourth to sixth months	227	7·6
First six months	487	8·1
Second six months	323	5·4

In Denmark it is recorded that whilst the mortality rate of whooping cough is decreasing the morbidity rate shows no decline. (Whooping cough is a notifiable disease in Denmark.) It may be considered that a guide to the number of cases of whooping cough in New South Wales is the report from the medical officer of the Department of Public Instruction, showing the number of cases of the infection notified by the teachers. Compared with this in Table VI are the South Australian figures (whooping cough is notifiable in that State). The cases notified to the Department of Public Instruction would cover only those children over five years of age.

TABLE VI.

Year.	South Australia.		New South Wales.	
	Cases.	Deaths.	Cases Notified to Department of Public Instruction.	Deaths for Whole of State.
1928 ..	574	21	2,923	103
1929 ..	497	1	10,933	212
1930 ..	4,466	38	5,483	164
1931 ..	495	6	9,022	186
1932 ..	1,542	4	3,214	61
1933 ..	1,008	11	1,533	25
1934 ..	1,313	7	19,030	286
1935 ..	3,616	23	3,813	63
1936 ..	751	9	10,024	120

The average fatality rate in South Australia for this period was 0·8%. Madsen quotes fatality rates for various age groups from Copenhagen as follows:

From 0 to 1 year	24·3%
From 1 to 5 years	2·6%
From 5 to 15 years	0·7%
Over 15 years	0%

(The years upon which these figures are based are not mentioned.)

From calculations based on the average fatality rate for South Australia for the years from 1928 to 1936 it is estimated that there must have been at least 35,000 cases of whooping cough in New South Wales in 1934.

It is estimated that about 78% of children have had whooping cough before reaching adult life. Collins[2] found that in six places in five different States of the United States of America the percentage of persons who had had whooping cough was within the limits 72·9 to 85·0. Stocks and Karn[3] (1932) showed that 44% of the children in London are attacked by the disease before the age of five years; they estimated that 61·0% have the disease during their life-time. From this it can be seen that the number of cases of whooping cough must be very considerable and that a child's chance of missing the infection must be very remote.

Probably no disease gives rise to a greater number of sequelæ. In a school or household it may be well nigh impossible to cope with the contacts satisfactorily.

As Stocks and Karn (1932) have very rightly pointed out, whooping cough has consequences that are much more serious than the measurable social significance of the infection; it may cause physical and psychic troubles of a permanent nature that cannot be reckoned in figures, such as alteration of the lung tissues and mediastinal lesions as revealed by X rays, and also probably nervous and cardio-vascular lesions, the ultimate consequences of which have not yet been determined. Finally, there is no doubt that the anxiety which precedes and accompanies the whoop may considerably disturb the emotional equilibrium of particularly delicate children.

Conclusion.

A brief review of some statistics has been made in order to demonstrate the importance of whooping cough as a cause of death in young children.

Acknowledgements.

My thanks are due to Dr. E. S. Morris, Director-General of Public Health, and to Dr. H. G. Wallace, Senior Medical Officer of Health, for helpful advice, and to the former for permission to publish these notes.

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Reports of Cases.

FATAL AND SEVERE HUMAN INFECTIONS WITH HÆMOLYTIC STREPTOCOCCI GROUP G (LANCEFIELD).

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Not many cases of fatal infection caused by hæmolytic streptococci of Lancefield's[1] groups other than Group A have been reported. Fry[2] (1938) recorded three examples of fatal puerperal Group B infections, two associated with a characteristic form of endocarditis.

Lancefield and Hare[3] (1935) isolated a Group G strain from the blood of a patient, from which they also obtained a heavy growth of staphylococci. The following case reports of fatal infections with Group G strains are therefore of interest.

Case I.

Clinical Record.

A woman, aged forty-four years, was admitted to the Royal Melbourne Hospital on August 13, 1939, as a patient of Dr. F. B. Lawton, complaining of malaise of one week's duration. She was very ill and unable to provide exact information regarding her symptoms. There was a history of measles and scarlet fever as a child, and of pleurisy twenty years before her admission to hospital. She complained of a slight cough accompanied by a small amount of sputum for three or four weeks. There had been no shivers or sweats, but some headache. She was flushed and cyanosed. Her *alæ nasi* were working. Her temperature was 104·6° F., her pulse rate was 112 per minute and her respirations numbered 30. The systolic blood pressure was 160 millimetres of mercury and the diastolic pressure was 100. The apex beat was felt in the fifth left intercostal space, three and three-quarter inches from the mid-line. There was one finger's breadth of right cardiac dulness. The cardiac rhythm was regular. There was a soft systolic bruit at the apex, and the aortic and pulmonary second sounds were accentuated. The percussion note over the lungs was resonant anteriorly, but it was impaired posteriorly at the apex of the left lung. The expiratory sounds were prolonged at both apices, the breath sounds at the apex of the left lung being almost tubular in quality. Crepitant râles were heard at the bases of both lungs, especially the right. No abnormality was noted in the abdomen. The calf of

the left leg was slightly reddened and noticeably hotter than the right, although there was no obvious source of infection. Both feet, in particular the left, were œdematous. No enlarged or tender glands were palpable. No phlebitis was detected. The ocular fundi were normal. The optic disks were slightly blurred at their nasal edges. The blood urea level was 84 milligrammes per 100 cubic centimetres. From the blood taken on the patient's admission to hospital Dr. Lucy Bryce cultured *Streptococcus anginosus*, that is, a hæmolytic streptococcus fermenting lactose but having no action upon salicin and mannite. (At this date the serological grouping of streptococci had not been introduced.) Two days after her admission to hospital there were definite physical signs of pulmonary consolidation at the base of the right lung. The inflamed area of the left calf was explored surgically, but no pus was found. The patient died two days later, that is, four days after her admission to hospital. Her illness was thus of about one month's duration. The findings at the autopsy performed by Dr. R. J. Wright-Smith were as follow.

Post Mortem Findings.

When the heart and pericardium were examined the sac was clear. The epicardium was normal except for a few small hæmorrhages along the lines of the coronary arteries. The heart was slightly enlarged, both ventricles being dilated. The myocardium was pale, soft and friable. There were a few subendocardial hæmorrhages. The *foramen ovale* was closed. The aortic, tricuspid and pulmonary valves appeared normal. The ring of the mitral valve admitted three fingers. The cusps showed no thickening of the valve. On the auricular surface, almost reaching the free border, were four small areas of ulceration, necrosis and softening. Superficial breaking down of the ulcerated areas had occurred; the process appeared to be fairly acute and there was no evidence of old endocarditis. The coronary arteries were patent and thin-walled and the orifices were clear. The aorta was normal in size and elasticity, and there was no interstitial abnormality. The renal, splenic, superior mesenteric and cerebral arteries were normal.

The right lung weighed 738 grammes (26 ounces) and the left 568 grammes (20 ounces). The pleural spaces contained no fluid or adhesions. Both lungs were greatly congested throughout, and numerous areas of bronchopneumonic consolidation, with large areas of surrounding œdema in the air-containing tissue, were present. They were friable. The bronchial mucosa was congested. The trachea and larynx were normal. The tracheo-bronchial lymph glands were not enlarged.

The skull and brain weighed 1,366 grammes (48 ounces). The *dura mater* was normal. The subdural space contained clear cerebro-spinal fluid. The pia-arachnoid over the hemispheres was somewhat œdematous and in its right frontal area there was an extensive subpial hæmorrhage. The substance of the hemispheres was swollen and pale, but otherwise clear. The basal ganglia, pons, medulla and cerebellum were normal, as were the ventricles.

The peritoneal cavity and lymph glands were normal.

The stomach was thin-walled and contained numerous terminal hæmorrhages and ulceration, without scarring. The remainder of the alimentary canal was normal.

The liver weighed 2,272 grammes (80 ounces). It was grossly enlarged, with a small surface and thin capsule. The liver parenchyma was intensely fatty and contained numerous hæmorrhages. Pronounced toxic spoiling was present. The whole organ was friable and greasy. The portal canals were clear. The gall-bladder was normal. The pancreas was swollen and congested.

The spleen weighed 395 grammes (14 ounces). It was enlarged to twice its normal size. On its postero-lateral aspect there were four fairly large reddish-white infarcts, quadrilateral in shape and surrounded by a zone of congestion. The splenic substance was intensely congested, soft, friable and pulpy. The suprarenals were normal.

The kidneys each weighed 170 grammes (six ounces). The capsules of both were thin and stripped readily. The right contained two fairly large yellow infarcts surrounded by a zone of congestion. Both kidneys contained numerous small hæmorrhages, probably embolic in character. The ureters and bladder were normal. The uterus was grossly enlarged and of globular shape. It was about six inches in length and contained a huge round interstitial fibromyoma, with a characteristic whorled appearance.

Case II.

Clinical Record.

A woman, aged forty-eight years, was admitted to the Royal Melbourne Hospital on August 19, 1938, under the care of Dr. J. B. Turner. Thirty-six hours prior to her admission the patient complained of soreness across the lower part of the back, together with shivers and fever. At the same time the right wrist became painful and the right arm was red and swollen. The redness subsequently spread to the axilla and the chest wall anteriorly and posteriorly. She had not noticed any previous cuts or infections on the hand or arm.

On examination a red, tender and raised area was seen, completely surrounding the arm and extending on to the axilla and to the chest wall anteriorly and posteriorly. The edge was sharply defined. No glands were palpable. The heart was not enlarged. Its rhythm was regular. There was a soft systolic murmur at the aortic area. The systolic blood pressure was 145 and the diastolic pressure 80 millimetres of mercury. The lungs and abdomen were normal.

A diagnosis of erysipelas was made and "Prontosil" was administered by mouth and intramuscularly. By the third day a very faint rash only was present over the affected area. The temperature, however, was 102° F. and the pulse rate 140 per minute. The following day the patient complained of pain in the chest and the physical signs suggested a commencing bronchopneumonia. On the fifth day she had further pain in the chest and pains of a severe character all over the body. A generalized skin rash appeared at this time. The patient's temperature and pulse gradually rose. From blood taken on the eighth day and incubated hæmolytic streptococci grew profusely. Her general condition deteriorated rapidly and death occurred on the tenth day.

Post Mortem Findings.

The significant findings at the autopsy by Dr. R. J. Wright-Smith were as follow.

The heart was normal except for the mitral valve, the cusps of which were covered with small, flat, yellowish, recent vegetations, with some ulceration of the posterior cusp. The vegetations extended on to the *chordæ tendineæ*. There was no evidence of old valvulitis. The lungs were slightly congested, but well aerated. In the lower lobes there were some areas of necrosis, but no consolidation or abscess formation was seen. The liver was swollen and friable. The spleen was swollen and greatly congested. There was a large recent infarct in the central area, with surrounding hæmorrhage. The kidneys were swollen and the surfaces hæmorrhagic; both contained numerous recent infarcts. There were small, raised, reddish patches about the lips. The erysipelas had disappeared.

Case III.

Clinical Record.

A woman, aged thirty-five years, about three months pregnant, was admitted to the Women's Hospital on May 28, 1929, as a patient of Dr. B. M. Sutherland, complaining of abdominal pain, vaginal hæmorrhage and rigors. She had a swinging temperature up to 102° F. during the daytime for four weeks after her admission to hospital. Her illness was diagnosed as septic incomplete abortion. The uterus was the size of a three months' pregnancy. Her heart and lungs were normal. From blood taken on the day of her admission to hospital and incubated a streptococcus was grown.

Two days after her admission to hospital 20 cubic centimetres of a 1% solution of mercurochrome were

injected intravenously. On the same day the fœtus and portions of placenta were aborted. A pelvic abscess developed; this was drained by posterior colpotomy on the sixteenth day. Drainage was not satisfactory and the incision was reopened on the twenty-third and again on the twenty-fourth day, when large quantities (about 20 ounces) of pus were liberated. No culture was made from the pus. An uneventful recovery followed. The patient was discharged on the forty-ninth day, her temperature having been normal for the preceding three weeks but for a transient rise to 100° F. for a period of three days.

Discussion.

The three strains isolated from the cases reported were sent to these laboratories from the respective hospitals at various times. Those from Cases I and III were obtained in 1929, and that from Case II was sent in 1938. Grouping was carried out by means of the formamide method of Fuller[a] (1938) for the extraction of the bacterial polysaccharide. The precipitin serum used was prepared against Griffith's[a] type XVI strain and checked against a serum prepared at the Bernard Baron Memorial Research Laboratories, Queen Charlotte's Maternity Hospital, London, kindly supplied to us by Dr. Lucy Bryce.

The clinical and *post mortem* evidence in Cases I and II indicates that the organisms cultured from the blood were responsible for the pathological changes. In Case III the evidence is not conclusive, but suggests that the organism grown played some part in the disease process.

From the clinical aspect, both fatal cases were typical bacteriæmias, associated with rashes or reddening of the skin. The *post mortem* changes were those usually found in bacterial endocarditis. In the first case the presence of the lesions on the auricular surfaces of the cusps of the mitral valve is somewhat unusual in view of the fact that no preexisting endocardial damage was noted. The absence of involvement of the posterior wall of the left auricle confirms the statement that there was no carditis prior to the onset of the fatal condition.

One feature of the endocardial lesions is of particular interest. The vegetations in two of Fry's cases due to Group B (non-fibrinolytic) strains were massive and crumbling. He suggested that this might be due to the absence of fibrinolysin, which would permit the heaping up of fibrin deposits, causing gradual enlargement of the vegetations. He described one case of endocarditis due to a Group A (fibrinolytic) organism, in which the lesions took the form of small, raised, localized plaques very different from the exuberant vegetations produced by the Group B organisms.

In both of our cases the morbid anatomy of the endocardial damage compares with that seen in Fry's Group A infection. The strain from Case I is non-fibrinolytic (but is ten years old and may have lost some of its earlier characters), while the other is weakly fibrinolytic, requiring 20 hours to produce complete lysis. There seems to be insufficient evidence at present to decide whether the fibrinolytic activity of strains is closely correlated with the type of vegetation.

The findings in the other organs are in the main similar to those seen in most cases of bacterial endocarditis. The wide dissemination of emboli producing multiple infarction without suppuration is the classical picture of this disease.

In both cases involvement of the skin and subcutaneous tissues was noted. In Case I it seems to be impossible to state that the lesion in the calf was primary, because of the preceding history of malaise of three or four weeks' duration. This history, together with the stage of development of the infarcts in both spleen and kidneys, suggests rather that the redness was secondary to the major lesion. Whether it was embolic in origin or due to circulating toxins cannot be determined.

In Case II the greater acuteness of the endocardial lesion and the freshness of the infarction suggest that the endocarditis was secondary to the erysipelas. The rash which appeared after the erysipelas cleared up suggests that Group G strains may produce an erythrogenic toxin, unless the rash was secondary to the "Prontosil" therapy. We have two other strains which were associated with scarlatiniform rashes. One was obtained from a patient with puerperal scarlet fever, and the other was grown in pure culture from the throat of a patient with mild scarlet fever. None of Griffith's original type XVI strains was isolated from a patient with scarlet fever. The patient who developed scarlet fever on the second day of the puerperium died after a stormy illness of five weeks' duration. The rash was bright and typical, and there was a pronounced enanthem on the palate. Unfortunately it proved impossible fourteen years later to trace the source of isolation of the streptococcus. It was probably isolated from the lochia, but possibly from a blood culture. It is unlikely that it was obtained from the throat. In view of this uncertainty the case cannot be regarded as a proved Group G infection. No autopsy was obtainable. There is some evidence, therefore, that Group G streptococci can produce rashes and erysipeloid infections. While rashes are almost always associated with Group A infections, Group C organisms have been isolated from erysipelas. Hare[a] (1935) mentions six examples of erysipelas due to Group *C* strains.

The bacteriological report on the organism from Case I as *Streptococcus anginosus* (Holman,[a] 1916) raises an interesting point. This strain still has the biochemical properties it possessed when first isolated in 1929. Two other Group G strains in our collection are similar. Sherman[a] (1937) has indicated that one type of Group G hæmolytic streptococci described by Bliss[a] (1937) has similar characters. This particular type, unlike our strains, belongs to the group of "minute hæmolytic streptococci". The interest of this possible relation between *Streptococcus anginosus* and Lancefield's Group G lies in the possibility that Group G strains may have been the cause of some of the fatal or serious infections attributed to *Streptococcus anginosus* in the earlier literature. Holman, for instance, obtained his *Streptococcus anginosus* on five occasions from the blood and on five from cases of cellulitis. It is probable that an increasing number of Group G infections will be identified now that Lancefield's methods are widely employed, and it should be possible to determine whether they are associated with a characteristic clinical and pathological picture.

Summary.

1. Three case reports of infection by organisms of Group G (Lancefield) are presented. Two of the patients died.

2. The characteristics of the chief pathological lesions found in the fatal cases, particularly endocarditis and inflammatory reactions in the skin, are discussed.

3. The possible relationship between Lancefield's Group G and *Streptococcus anginosus* (Holman) is briefly considered.

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SIMULTANEOUS SEPTICÆMIA AND PERITONITIS CURED BY SULPHANILAMIDE.

By ALAN GRANT,

WITH A COMMENTARY BY

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Clinical Record.

(Alan Grant.)

Mrs. M.J., a *primipara*, aged twenty-five years, had suffered from "influenza" for four days. She was confined in a maternity hospital in the suburbs on May 8 at 2.20 a.m. The pregnancy was at full time and the baby was born alive in what was described as "a normal confinement", except that the patient sustained a second degree tear. During that day she appeared well. At 4 p.m. her temperature was normal. At midnight she complained of an increasingly severe abdominal pain. She was sent to the Women's Hospital, Crown Street, the next afternoon (May 9) at 2 p.m.

On examination her temperature was found to be 40° C. (104° F.). Her pulse rate was 148 per minute, and respirations numbered 48 per minute. She looked gravely ill. Her lips were slightly cyanosed, the tongue was dry, and sordes were present. The patient was not vomiting and there was no history of vomiting. All systems were clear except for the abdomen. It was distended and tender all over, but there was no rigidity and there were no masses except the *fundus uteri* at a normal level. *Per vaginam* I found a second degree tear and no further abnormality. The provisional diagnosis of peritonitis was made and the following investigations were carried out: a cervical swabbing and a catheter specimen of urine were examined, an attempt was made to culture organisms from the blood and a swab from the throat was examined.

Treatment.

At once four grammes of sulphanilamide were given (eight tablets); one gramme (two tablets) was then ordered to be given every four hours (five times a day).

The patient was placed in Fowler's position and copious fluids were given by mouth.

At 9 p.m. on the same day her condition was unimproved. Her temperature was 38·8° C. (101·8° F.), her pulse rate was uncountable over 150 per minute. The respirations numbered 46 per minute. We decided to operate at once.

The patient was placed almost flat in the ward bed and her bladder was catheterized. Instruments and spotlights having been arranged, the abdomen was opened in the mid-line suprapubically under local anæsthesia, supplemented at one stage with a small amount of ether given by the open method. Large quantities of thin blood-stained pus exuded. Three long drainage tubes were inserted so that one drained each paracolic gutter and one went into the pouch of Douglas. (This procedure is not thought to be ideal. Two flank incisions and a mid-line incision would be better. However, the primary issue here was to save the patient much shock. These tubes caused colic later. *Vide infra.*)

The pus was found by examination of a direct smear to consist of Gram-positive cocci, mostly in pairs, but a few short chains were seen. They were almost universally intracellular.

On the second day (May 10) the patient's temperature was 38·4° C. (101° F.), her pulse rate was 148 and her respirations numbered 38 per minute. Her condition was worse; her lips were cyanosed and her abdomen was more distended. The tubes were draining unequally; they were turned and shortened. This procedure was followed by a good flow of pus.

The results of the investigations were by this time available and were as follows: the attempt at culture from the blood had resulted in a profuse growth of hæmolytic streptococci; the cervical swab had been found to contain hæmolytic streptococci; hæmolytic streptococci were present in the pus from the peritoneal cavity; a centrifuged deposit of the urine showed an occasional pus cell. An attempt at culture on McConkey's medium resulted in no growth of organisms. A blood count revealed that the red blood cells numbered 3,720,000 per cubic millimetre, and the hæmoglobin value was 55%. The leucocytes numbered 4,300 per cubic millimetre; 80% were neutrophile cells, of which 28% were segmented and 52% were band forms; 8% were lymphocytes, 3% were monocytes and 9% were myelocytes. The leucocytes had a pronounced shift to the left. Hæmolytic streptococci were present in the throat swab. The patient's blood group was Type III.

Here, then, was a patient suffering from first-day puerperal septicæmia, together with streptococcal peritonitis. Large doses of sulphanilamide were indicated; but her blood count revealed a leucopenia. A small dose of pentnucleotide was given.

On the third day of illness, May 11, the patient's temtemperature was 38·4° C. (101° F.); her pulse rate was 145 and her respirations numbered 30 per minute. Her general condition was slightly improved, but she was still blue. Her abdomen was still distended, but there was no vomiting. At 12 noon a spectroscopic examination of the blood revealed a very small amount of methæmoglobin present, but no sulphæmoglobin. Her blood sulphanilamide level at this time was 8·7 milligrammes *per centum*. This was below the desired level, so the doses of both pentnucleotide and sulphanilamide were increased. The patient was given saline solution by the continuous intravenous drip method, an intramuscular injection of eight cubic centimetres of pentnucleotide, 0·5 cubic centimetre of pitressin every four hours, and 1·5 grammes (three tablets) of sulphanilamide every four hours—a daily total of 7·5 grammes. She was also given a blood transfusion. During this day 1,600 cubic centimetres of 10% glucose in saline solution were given, together with 575 cubic centimetres of citrated blood. The bowels acted well during the afternoon and the tubes in the abdomen were again turned and shortened.

On the fourth day of illness, May 12, the patient's temperature was 38·2° C. (100·8° F.); her pulse rate was 120 and her respirations numbered 26 per minute. Her general condition was only fair. She was complaining of intermittent abdominal pain, so the tubes were further shortened and turned. At 11.30 a.m. the blood sulphanilamide level was 9·3 milligrammes *per centum*, a concentration of almost 1 in 10,000. The peritoneal fluid was also subjected to an estimation of sulphanilamide content and was found to contain 10·7 milligrammes *per centum*. It will be seen that the blood sulphanilamide level was reaching an optimum figure. A leucocyte count revealed only 5,200 per cubic millimetre.

For these reasons the patient received the following treatment on the fourth day: (i) a second blood transfusion of 525 cubic centimetres of citrated blood; (ii) ten cubic centimetres of pentnucleotide; (iii) one gramme (two tablets) of sulphanilamide every four hours, five times a day.

On the fifth day of illness, May 13, the patient's temperature was 37·8° C. (100° F.). Her pulse rate was 130

and her respiration rate 20 per minute. She was slightly improved, though still blue and becoming somewhat breathless. The distension of the abdomen was reduced considerably and her bowels were acting freely. Two of the tubes draining the abdomen were again shortened and turned, and the one into the pouch of Douglas was removed altogether. The treatment of this day consisted of 12 cubic centimetres of pentnucleotide and doses of sulphanilamide of 0·5 gramme and 1·0 gramme alternating every four hours.

On the sixth day of illness, May 14, the patient's temperature was 37·7° C. (99·8° F.); her pulse rate was 126 and her respiration rate 20 per minute. She was much better and the drainage tubes were removed completely. The wound was strapped.

On the seventh day of illness, May 15, the streptococci causing the condition were reported to be Lancefield group A, type XI (Griffith). (The streptococci in the cervix, blood, peritoneal cavity and throat were identical serological types.) A blood count was made; the erythrocytes numbered 3,970,000 and the total leucocytes 13,700 per cubic millimetre. The hæmoglobin value was 68%.

On the eighth day of illness, May 16, the patient was further improved and the dosage of sulphanilamide was reduced to 0·5 gramme every four hours. From this day onward steady progress took place until the eleventh day, when the sulphanilamide was suspended.

On the morning of the twelfth day the patient complained of a pain in the right side of the chest, commencing during the night. Her temperature was 38·9° C. (102° F.); her pulse rate was 114 and her respiration rate 31 per minute. At the base of her right lung there had suddenly appeared dulness, coarse crepitations and tubular breathing. The patient was given oxygen and morphine, and also one gramme of 2-sulphanilyl-amino-pyridine ("M & B 693") every four hours. The signs were consistent with the presence of a pulmonary infarct. During the next three days the chest complication rapidly subsided.

Subsequent to this setback the patient had an uninterrupted recovery. On the twenty-second day of the illness she received a third blood transfusion. About the middle of June she left hospital with her infant. At a future date she will need a plastic operation on the perineum, where the second degree tear had healed unsutured.

It should be mentioned in conclusion that the baby contracted pharyngitis during the mother's illness, and this was found to be of streptococcal origin also.

Commentary.

(John Chesterman.)

The case history of this patient, whom I was fortunate enough to see throughout her illness in collaboration with Dr. Grant, illustrates well many points in our present methods of treating such an infection. The patient was suffering from an early fulminating puerperal infection of the peritoneal cavity and blood stream by the *Streptococcus pyogenes*, the very possible source of the organism being her own respiratory tract. Before the discovery of sulphanilamide, this combination of peritonitis and septicæmia was invariably fatal, and it is probable that even sulphanilamide would not have overtaken the infection in this case without the liberation of the pent-up toxic fluid within the peritoneal cavity. The effect of peritoneal drainage itself is shown by the following table, published before the introduction of sulphanilamide, showing the results obtained in the isolation block of Queen Charlotte's Hospital.

TABLE.[1]

Peritonitis. The Effect of Drainage.

	Deaths.	Mortality Rate.
First series: 31 cases:		
14 Drained—often late	13	93%
17 Undrained	17	100%
Second series: 22 cases:		
18 Drained—often early	11	61%
4 Undrained	4	100%

[1] Taken from *The Proceedings of the Royal Society of Medicine.*

This same report records 28 cases in which *Streptococcus pyogenes* (hæmolytic) was cultured from the genital tract, blood and peritoneum, with a case mortality of 100%. It is probable that, with proper use, sulphanilamide will save from operation the patient with commencing generalizing peritonitis; but where the whole peritoneal cavity is involved at the time the patient is first seen, in our opinion drainage should be adopted as well. As this case illustrates, this can and should be carried out with as little disturbance to the patient as possible. The proper use of sulphanilamide in a severe infection means the attainment and maintenance of a concentration in the blood of at least 10 milligrammes *per centum* (that is, 1 in 10,000). The concentration in the blood may be estimated by the colorimetric test devised by Marshall, Emerson and Cutting,[(2)] and it is necessary to make this estimation because there is some individual variation in the dosage of sulphanilamide required to reach the desired concentration. In my experience the average dose necessary for this is about 10 grammes in the first twenty-four hours and five to six grammes in each succeeding twenty-four hours. It should be remembered that these doses are recommended for the more severe generalized infections. It is wise to continue sulphanilamide, in reduced doses, for five days after the temperature has fallen to normal. Failure to do this has in several instances been followed by a relapse, the rapid elimination of the sulphanilamide apparently allowing the organisms to commence multiplying again. Since sulphanilamide, like urea and alcohol, is readily diffusible through the body fluids, the concentration in the peritoneal exudate (10·7 milligrammes *per centum*) was of a similar order to that in the patient's blood.

In spite of our patient's pronounced cyanosis, spectroscopic examination of the blood revealed only a small amount of methæmoglobin and no sulphæmoglobin. It was safe to ignore the cyanosis, and indeed this holds in most cases in the absence of other signs of toxicity. The action of the sulphanilamide on the streptococci is not bactericidal, but bacteriostatic; the organisms are not killed, but their reproduction is hindered, and the natural defence mechanisms of the body thus have a chance of destroying them. It follows that all possible measures to aid the body's attacking powers must be undertaken. When this patient arrived at hospital her leucocytes had been reduced to 4,300 per cubic millimetre (a smear from the peritoneal fluid was crowded with leucocytes, most of which contained streptococci) and the differential leucocyte count revealed a pronounced "shift to the left", indicating the attempt of the bone marrow to meet the demand. After her first blood transfusion and the slowing down or cessation of bacterial reproduction the leucocyte count rose to 5,400 per cubic millimetre. In three days, during which a second blood transfusion and intramuscular injections of pentnucleotide were given, the leucocytes increased to 13,700 per cubic millimetre. With the invading organism held in check, then, these would seem valuable means of stimulating the body defence mechanisms.

Acknowledgements.

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Reviews.

RECENT PROGRESS IN MEDICINE.

As is to be expected, the latest edition of the popular "Beaumont and Dodds" contains much new material of value to the clinician and laboratory worker.[1] Such is the growing importance of the sulphanilamide drugs that the authors have found it necessary to devote the whole of the opening chapter of the book to a concise yet very complete discussion of their chemistry and clinical applications. The section on vitamins has been brought up to date by the inclusion of references to the recent work of Ansbacher, Thayer and others on vitamin *K*.

The treatment of urinary infections by ketogenic diets, which was fully considered in the previous edition, has now been omitted, though the authors admit that it marked a great step forward in the therapy of these conditions. Instead, tribute is paid to the worth of treatment by means of mandelic acid, especially in uncomplicated cases of pyelitis and cystitis. A great deal of revision is apparent in the section on the dietetic control of diabetes; the fresh matter includes entirely new analyses of foodstuffs by Widdowson, Shackelton and McCance. From the purely clinical standpoint some of these tables appear to be a little too elaborate. There are also statements concerning the use and action of protamine zinc insulin which will not be accepted unreservedly by many physicians.

A new series of electrocardiograms, supplied by Dr. D. E. Bedford, shows the value of chest leads in the diagnosis of coronary disease. In Chapter X a summary is made of all recent work on the sex hormones, with special emphasis on the clinical use of these substances; mention is also made of the anterior pituitary hormones and of the latest research on the cortical hormone in Addison's disease. The field of investigation offered by the introduction of the gastroscope is also the subject of comment for the first time.

A fair criticism of this book is that while it has been said by some to lay stress upon "test-tube" rather than upon "bedside" investigation, yet its generally comprehensive viewpoint and its authors' ability to sift the chaff of medicine from the grain give it a unique position among all that is best in modern scientific literature.

OPERATIVE SURGERY.

"ROYAL NORTHERN OPERATIVE SURGERY", published by H. K. Lewis and Company Limited, has been written by eleven surgeons of the Royal Northern Group of Hospitals and is designed to represent the surgical procedures found to be most satisfactory by the authors.[2]

The general impression is that this is not a book for either students or the occasional surgeon, but for the trained surgeon in busy practice. The subject matter is condensed to essentials and presupposes an intimate knowledge not only of anatomy but of general surgical technique. As the operations described are measures well tried by capable surgeons, but little criticism can be offered other than in certain particulars.

Parotid tumours are indicated as being of frequent recurrence, yet the procedure given is enucleation of the tumour and then excision of its capsule.

In regard to tuberculous glands of the neck the authors' practice is not to attempt anything in the nature of a block dissection, "as the accessory nerve must perforce be divided". This practice has not been adopted in other clinics, and, if correct use is made of fascial planes, Koenig's comment that "in such cases the operator needs iron patience and plenty of time" will not apply, nor will he need to be seated for the operation.

A two-stage operation is used for pharyngeal diverticulum, but modern practice now favours the one-stage operation with the help of a colleague using the œsophagoscope.

It is surprising to find a suprahyoid dissection of the neck advised "for the type of case where common sense dictates it, *viz.*: cases of carcinoma of the lower lip, the floor of the mouth and in malignant disease of the submaxillary gland". Thus the essential deep cervical glands are left untouched. However, a very radical block dissection is described, and although this is stated to be unilateral, the remark is made that it is illogical to perform a very extensive operation on one side and to do nothing on the other.

With thyreoidectomy it is obvious that the fascial relations of the gland are disregarded, as evidenced by reference to the sterno-thyreoid muscle, which, "if well defined, can be retracted but there is no particular reason to spare it"; and again, "difficulty in exposing the upper pole is usually due to failure to free the sterno-thyroid fibres clinging to the gland".

Good advice is given in regard to the development of technique by the use of local anæsthesia. In reference to total thyreoidectomy the ingenuous remark is made that "it is purely an anatomical dissection". Surely this applies to most surgical technique!

The section on thoracic surgery is a very good résumé of the subject, and the section on abdominal surgery is probably the best feature of the book.

In hydatid of the liver stress is laid on the separation of the ectocyst from the endocyst, and this does not seem to connote a very great familiarity with the disease.

It is stated that the Harris operation for prostatectomy "has not met with the success that its originator anticipated" and must be regarded as a stepping stone in the history of prostatic surgery. This is not in accord with the experience of those who have kept strictly to the details of the technique as laid down by the late Harry Harris, as any deviation leads to disappointment.

A comprehensive survery of modern practice in orthopædic surgery is given. In the chapter on fracture of the neck of the femur, however, the Fox apparatus is mentioned and pictured, and then at the end it is pointed out that the apparatus has not been manufactured commercially.

The volume concludes with brief mention of ligature of the important arteries, the technique of blood transfusion and the treatment of varicose veins.

On the whole the book is well illustrated and the publishers have done their part in excellent fashion.

NEW LIGHT ON OBSTETRICS AND GYNÆCOLOGY.

THE appearance of the fourth edition of "Recent Advances in Obstetrics and Gynæcology" will be hailed with pleasure by the busy practitioner and specialist alike.[1] This new edition is much reduced in the first portion devoted to obstetrics; but additions are chapters on pyelitis and on breech deliveries, and an appendix devoted to the active principles of ergot. The second part, devoted to gynæcology, has been much expanded, notwithstanding that the chapters on endometrioma and functional uterine hæmorrhage have been omitted. The new knowledge of the sex hormones is allotted a chapter of twenty pages, and a welcome addition is a chapter by Wilfred Shaw on the histogenesis of ovarian tumours. New also is the chapter given to leucorrhœa, in which Dr. Bourne takes the opportunity of describing his own technique in the treatment of cervicitis with zinc chloride. The chapter on physical therapy in gynæcology, again contributed by Justina Wilson, has been largely rewritten so as to be brought into line with recent developments and improvements.

[1] "Recent Advances in Medicine: Clinical, Laboratory, Therapeutic", by G. E. Beaumont, M.A., D.M., F.R.C.P., D.P.H., and E. C. Dodds, M.V.O., D.Sc., Ph.D., M.D., F.R.C.P.; Ninth Edition; 1939. London: J. and A. Churchill Limited. Large crown 8vo, pp. 447, with 42 illustrations. Price: 15s. net.

[2] "Royal Northern Operative Surgery", by the Surgical Staff of the Royal Northern Hospital; 1939. London: H. K. Lewis and Company Limited. Super royal 8vo, pp. 564, with 463 illustrations. Price: 42s. net.

[1] "Recent Advances in Obstetrics and Gynæcology", by A. W. Bourne, M.A., M.B., B.Ch., F.R.C.S., F.R.C.O.G., and L. H. Williams, M.D., M.S., F.R.C.S., F.R.C.O.G.; Fourth Edition; 1939. London: J. and A. Churchill Limited. Large crown 8vo, pp. 376, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 23, 1939.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE WAR AND AUSTRALIAN MEDICINE.

ONCE more Britain is at war, and Australia with her. How valiantly the ministers of State in the Old Country strove to keep the world at peace we all know. We have all for many months past followed the happenings in Europe; sometimes a ray of light has come, to be obscured again; even in the last days we hoped with millions of our fellow men that a way out would be found; and we must agree with the Prime Minister of Great Britain that, had reason prevailed, a way could have been found. No one can have listened unmoved to the words of Mr. Chamberlain when he spoke on the air from Number 10, Downing Street, or of His Majesty the King when he spoke a little later to the people of His Empire. These words were a call to duty—to service, to self-sacrifice and to untiring effort—and we may be sure that no Australian will fail to answer the call.

Medical men and women have a dual role. As citizens of Empire they will oppose the enemy, throwing all their energies into the fight, keeping their hands clean and their heads clear, and hating only the oppression, the cruelty, the madness and the persecution which have brought this dire calamity upon the world. As doctors they belong to a branch of science which, as such, has nothing to do with the causes of war, and which is not concerned (as was stated in this journal in August, 1914) with the justice or wisdom of strife. Medicine knows neither nation nor race; and the medical profession has but one aim, to prevent disease and to cure it wherever and whenever it occurs. "No sacrifices are too great for the profession in its continuous struggle against trauma, disease and death." Though the two roles of medical man or woman cannot be separated, their recognition is of more than academic importance. They give to the follower of medicine a breadth of outlook that is denied to many in other callings; they endow, or should endow, him with a sense of values different from that of other people; and they clothe him with responsibilities that are peculiarly his. In the last war of 1914 Australia gave freely of her manhood and the medical profession took its share of the burden, many of its members giving their lives in service overseas. Medical men and women as individuals will again offer their services to the State; but there is another aspect, that of organized medicine. At its meeting on September 6, 1939, the Federal Council of the British Medical Association in Australia adopted a resolution expressing the willingness of the Association to cooperate with the Commonwealth Government in the defence of Australia. Though this willingness might justly have been assumed, its formal expression was valuable; and the public may rest assured that the Association will by every means in its power cooperate with the authorities in providing medical attention not only for the fighting forces but for the civil population. Already members of the Association are serving on the coordination committees which were mentioned in these pages last April. It is to be hoped that the authorities, when they are calling to the colours even the Army Medical Corps officers on the active list, will take advice from these committees. The best possible use must be made of available medical men, and

to this end the attainments of the officer (or medical practitioner) concerned should be considered. Not only should these committees be asked for advice; they should also be allowed to draw up schemes for submission to the authorities. An unsuitable scheme can always be rejected, particularly if it is unworkable in the light of information available only to official circles.

We have stated that the best possible use must be made of available medical men. This is not an idle remark. During the last war many a square peg was jammed into a round hole in the overseas medical services, particularly in the early stages of the war. In these circumstances the highest efficiency cannot be maintained. In the present conflict attention must be paid to the needs of the civil population, equally with those of the fighting forces. This applies particularly to the specialist services of the large public hospitals, both in city and country districts. If each practitioner is regarded merely as another individual in the general medical services, and not from the point of view of his professional skill, it is easy to visualize a depletion of the members of a single specialty in a public hospital staff. The civil population would then be deprived of necessary services. By careful planning such an unfortunate state of affairs can be avoided. This planning can be carried out by the State coordination committees, and members of the specialist staffs will be found ready and willing to cooperate.

Current Comment.

THE ELIMINATION OF GIARDIA INFECTION.

INFECTION with *Giardia lamblia* may occur in any part of the world, but is commoner in tropical countries. The organism, a flagellate protozoon, inhabits the lumen of the small intestine. There has been some controversy concerning its pathogenicity. Many observers declare that it is non-pathogenic, and point out that the organism in its encysted form is often found in the stools of persons apparently in perfect health. Others are convinced that it is responsible for such intestinal disturbances as diarrhœa, abdominal pain, flatulence *et cetera*. It is of interest to note here that giardia has been found in the stools of children suffering from cœliac disease, and a causal relationship has been suggested. At any rate, most people who know themselves to be infected would be glad of the knowledge of some means for the elimination of the infection. Various drugs have been employed, mostly with indifferent success. Recently, R. N. Chopra, B. M. Das Gupta, B. Sen and Z. Ahmed have given "Atebrin" to patients harbouring the parasites.[1] They were prompted to investigate the therapeutic properties of "Atebrin" by the report of experiments carried out by Brumpt and the work of Martin in 1937. Brumpt "conducted experiments on lamblia-infected mice and has noted that, of all the treatments tested, oral administration of one per cent solution of quinacrine (a French equivalent of atebrin) for 5 days was able to bring about a cure in 80 per cent of the animals to which it was administered". Martin noted that the administration of "Atebrin" in the doses employed in the treatment of malaria cured the majority of patients of their giardia infection.

Chopra, Das Gupta, Sen and Ahmed treated a number of patients by the oral administration of "Atebrin" in a dose of 0·1 gramme (one tablet) three times a day for five days. They present the records of ten patients whom they were able to keep under observation for six weeks after treatment. In every case the parasites disappeared from the fæces after nine tablets of "Atebrin" had been given, and, in those cases in which a period of observation was possible, they did not reappear.

In discussing the possible pathogenicity of giardia they point out that rats and mice are frequently found to be infected with *Giardia muris* and that *post mortem* examination of these animals reveals no lesions of their intestinal tracts. The organism lives in the fluid contents of the gut and does not penetrate the mucosa. On the other hand, the fact that healthy persons may harbour the parasite does not prove that it is non-pathogenic; for such a dangerous organism as *Entamœba histolytica* is found in the stools of a moderate percentage of apparently healthy people. It has been stated by some observers that children infected with giardia are apt to suffer from "intractable diarrhœa associated with the passage of large quantities of mucus and impairment of growth as a result of chronic indigestion". Some have even expressed the opinion that the organism can be responsible for cholecystitis. In the series of ten cases investigated by Chopra and his collaborators, three patients had no intestinal disturbance; two were infected with *Entamœba histolytica*, which was thought to be responsible for their symptoms; the remaining five suffered from diarrhœa or other intestinal disorder and their symptoms diminished or disappeared with the eradication of the infection. Chopra and his collaborators express the opinion that "in some cases at least infection with *Giardia lamblia* is responsible for intestinal disturbances, especially in children". They point out that while "Atebrin"

[1] *The Indian Medical Gazette*, August, 1939.

is invaluable in the eradication of giardia, it has no action against any of the other flagellates or *Entamœba histolytica* or *Entamœba coli*.

We are inclined to agree with the opinion of these workers that *Giardia lamblia* can cause functional disturbances of the intestine, hence, directly or indirectly, serious illness. Endeavours should be made to eradicate the infection. The administration of "Atebrin" would appear to be the most efficient means of achieving this result.

ALLERGY GOUT.

Gout is a condition which lies in a medical backwater, only occasionally disturbed by the currents of minor controversy concerning its chemical aspects. Thought is rather like an invisible man who must be clothed by language to be perceived by others; but this garment of thought sometimes obscures its outlines with considerable thoroughness, even in scientific circles. Thus gout at one time was a blessed Mesopotamia of a word, covering a multitude of conditions, just as the wide, or perhaps we might say delightfully wide, expressions "error of metabolism" and "endocrine disturbance" fulfil a similar handy function today. Garrod began our exact chemical knowledge of the uric acid disturbances which are so striking a feature of this disease, and the last word has not been said yet. It is not difficult to understand the mechanical variety of gouty attack—such may sometimes be seen after radiation therapy in leuchæmia, for example—nor is it hard to believe that at the other end of the scale some of the cutaneous manifestations once considered to be of gouty origin may be allergic in nature.

W. J. Stewart McKay has attempted to assemble these two extremes in one concept and writes of what he calls "allergy gout".[1] His contention is that some persons born with the gouty diathesis have not only difficulty in metabolizing purins and excreting their products, but also may exhibit symptoms of allergy. He further contends that the possession of the allergic constitution may modify the manifestations of gout, especially in later life, thus explaining the occurrence of what some textbooks describe as irregular forms of gout. McKay gives detailed accounts of many of the traditional and familiar signs and symptoms which herald, announce and follow a gouty paroxysm. He then quotes numbers of conditions frequently observed in allergic persons, and attempts to analyse the gouty and the allergic factors in the so-called irregular gout, or, as he would call it in one special variety, allergy gout. Certainly the occurrence of asthma and migraine and other manifestations which may be allergic has often been noted in gout. McKay states that when a gouty diathesis has been proved it is then necessary to inquire also into the possibilities of allergy in the patient, when presumably a diagnosis of allergy gout would be made, though he remarks that some persons are more gouty than allergic, and others more allergic than gouty. We cannot help having a suspicion that this descriptive nomenclature amounts to little more than reshuffling the titles to the pigeon-holes and rearranging their contents; yet there may be something in the idea that the allergic diathesis may modify the manifestations of gout. The real crux lies in the answer to the question, "what is gout?" Does it follow that all the symptoms attributed to gout are really due to this metabolic dyscrasia even in cases in which major seizures undoubtedly occur? In assuming this we are in danger of swinging back to the firmer but blinder faiths of two generations ago. What we really want to know is what underlies the production of gout; if we had surer knowledge of this we should then be in a better position to say positively what symptoms and signs could truly be attributed to this cause. Until this can be done with certainty we are not much ahead of those who described "irregular gout"; and if McKay's notion of a combined ætiology in certain cases does not help us far along this road, at least it brings to our attention a subject which today is often forgotten.

[1] *The Medical Press and Circular*, May 31, June 7 and 14, 1939.

THE DRUG TREATMENT OF HYPERPIESIA.

"The urge for trying new remedies in hyperpiesia (essential hypertension) is prompted by the common incidence of a condition which produces bizarre symptoms and determines many complications." William Evans and Owen Loughnan thus commence a record of their findings in regard to the relative value of many drugs in the treatment of no less than 70 patients suffering from essential hypertension. Females preponderated considerably, and the average age of the whole group was fifty-eight years. The minimum requirement of a patient's blood pressure before his or her admission to the series was fixed at 160 millimetres of mercury for the systolic and 100 millimetres of mercury for the diastolic pressure. The patients received a careful initial clinical survey, and all nephritic persons were excluded. Over a thousand estimations of blood pressure under standard conditions were made in a period of eighteen months. The patients were seen at least every fortnight, and many were admitted to hospital. The drugs were administered in adequate and usually in optimum doses. Before a remedy was judged to have established a claim to be of value as a hypotensive agent it had to satisfy three standards. It had to be capable of maintaining a reduced blood pressure; this reduction had to be consistent in a high proportion of patients; and finally, this reduction had to be maintained without the production of any toxic symptoms.

A rise or fall of less than ten millimetres in the systolic pressure was not taken into consideration. Altogether no less than 33 remedies were tried,

including many proprietary preparations, nitrites, xanthine derivatives, vegetable extracts, sedatives, iodine, choline derivatives and dietetic restriction. Some of these are worthy of more detailed description. The effect of nitrites upon blood pressure in healthy and hypertensive persons was carefully examined by Weiss and Ellis in 1933. The present authors have substantially confirmed their findings. The fall following inhalation of amyl nitrite was transitory, averaging three minutes, and continued longer in the patients with hyperpiesia than in normal persons. The extent of the lowering of systolic pressure averaged 45 millimetres. A compensatory rise of blood pressure to a slight degree was observed in only four of 22 persons. Glyceryl trinitrate produced comparable, if slightly later, effects and a more prolonged depression (an average of sixteen minutes). Both drugs are therefore depressor, but unlikely to prove of value for continuous treatment of hyperpiesia. In controlled test periods, each of fourteen days, sodium nitrite, erythrol tetranitrate, mannitol hexanitrate, and bismuth subnitrate were employed, with inconclusive results. Iodine, as Lugol's solution, and potassium iodide failed to convince the authors that they possessed any hypotensive qualities. Interesting results were obtained with the sedatives, bromides, "Sodium luminal" and chloral hydrate. No regularity of fall was seen, and in some instances a rise occurred. Even symptomatic improvement was highly inconsistent. The xanthine derivatives "Euphyllin" and "Diuretin" were no more successful or constant in effect; an approximately equal number of persons showed slight blood pressure fall to those whose pressures remained stationary or even rose slightly.

The more stable acetylcholine derivatives "Doryl" and "Mecholin" are active when given by mouth. Evans and Loughnan arrived, however, at the same unfavourable conclusion as Fraser, namely, that they were valueless for the treatment of hypertension, except in the relief of hypertensive headache. Similarly, calcium salts, atropine, potassium thiocyanate and benzyl benzoate produced no change or produced a rise in blood pressure just as often as a fall. No toxic effects were seen after the thiocyanate. "Guipsine" and "Detensyl" (extracts of mistletoe and viscera), "Phyllosan", "Citrin", "Yohimbine", "Padutin", all failed to give any evidence of therapeutic value in the treatment of hyperpiesia. Even substances such as anabolin, œstrone and "Perandren", of proved experimental activity, quite failed to pass the strict control standards demanded by the authors.

Employment of reducing diets as a means of lowering high blood pressure in obese individuals is an accepted practice. Evans and Loughnan, after watching the effects of weight loss upon the blood pressure, conclude that the results are no more than can be explained by "natural variation of blood pressure", although symptomatic improvement from loss of weight usually took place. They state that as this extensive investigation proceeded they came to realize more fully how great is the natural variation and how false would be the judgement of the hypotensive action if this were ignored. They have refrained from crediting any hypotensive property to any drug unless it has exerted this effect to a greater extent than that which occurs naturally during a period on *placebo* treatment. Neglect of this precaution would appear to have led other investigators into error. Another common fallacy apparently is to accept the initial (that is, the very first) reading as the average normal for the individual, whereas in point of fact subsequent study shows it to have been spuriously high. The extent of fall before a basic level was reached in such emotional persons was often over 25 millimetres and was not complete for at least 15 minutes.

Evans and Loughnan's work is valuable. It points again the moral of accepting with considerable scepticism clinical promises based upon pharmacological findings in laboratory animals, and constitutes a brilliant arrow indicating the absurd but prevalent fallacy of forming conclusions upon small and uncontrolled personal experience.

TUBERCULOSIS ENDOTOXOID.

THE specific treatment of tuberculosis dates from the year 1890, when Robert Koch announced his discovery of tuberculin and all the world was fired with the hope that a sovereign remedy was found at last.

> In spite of the quackish and quizzical,
> May Koch's magic lymph anti-phthisical
> Effect a safe cure,
> As lasting as sure,
> O'er the saddest of maladies physical.

This jingle from the preface to a contemporary volume of *Punch*, which was dedicated to Koch, crystallized the popular feeling of the day. But the untoward effects which followed and still follow the incautious use of tuberculin, and the failure to achieve anything but disappointment by many who possessed the background, the capacity and the zeal for mastering its possibilities, notwithstanding the wonderful claims made for it by a few, equally well qualified to speak, who still regard it as a sovereign remedy, have made it the subject of violently partisan controversy for long years, and the end result has been its virtual disappearance from the orthodox *materia medica*. Some of the best results of treatment with tuberculin have been claimed in extrapulmonary tuberculosis; but the now wide recognition that disseminated tuberculosis may exist in a benign and non-caseating form must lead to a review of some of these claims. Specific treatment of tuberculosis by means of tuberculosis antiserum and by vaccines has often been attempted and encouraging results have been reported; but that is all. No serum or vaccine has yet succeeded in establishing a place for itself in the orthodox treatment of tuberculosis. The multiplicity of different

kinds of tuberculin and of serum and vaccine, each with its own group of exponents, has produced some bewilderment in the mind of the profession at large and has emphasized our imperfect understanding of the complexities of the mechanism of resistance and immunity to tuberculosis. Hence general and heavily armoured scepticism when a new form of specific therapy is announced is only to be expected. Nevertheless a new form of specific therapy, which has been called tuberculosis endotoxoid, prepared in 1932 by E. Grasset, of the serum department of the South African Institute for Medical Research, appears to merit more than passing attention. The mode of preparation, the chemistry and the immunological properties in animals and preliminary results of its therapeutic application to human subjects suffering from tuberculosis have been reported in the European literature from time to time in recent years; there now appears in the English language[1] an account of the treatment of 242 persons, inmates of eight State institutions for tuberculosis in South Africa, under the supervision of the respective institutional medical officers.

Briefly, tuberculosis endotoxoid consists of the antigenic principles obtained from tubercle bacilli by an extraction process of repeated freezing, activated by means of a hydrolysed peptic medium and detoxicated by the action of formalin. The product is atoxic and stable; it contains the water-soluble lipoid fraction of the tubercle bacillus, has a low bacillary protein content and can be injected in doses of several cubic centimetres without the production of toxic or sensitizing phenomena in normal or tuberculous subjects. In his earlier papers Grasset claimed that the administration of tuberculosis endotoxoid in progressively increasing doses exerted a definite therapeutic action on the evolution of tuberculous disease, the systemic and organic improvement being verifiable by clinical, functional, radiological and bacteriological tests.

Of the 242 persons whose treatment with endotoxoid is described in Grasset's latest paper, 136 were Europeans suffering from pulmonary tuberculosis, and most of them were in an advanced stage of the disease; of these, 87 were benefited. It is stated that even when the lesions are extensive and advanced a double healing process is usually to be observed in the lungs within a few months, consisting of a clearing of the more recently affected peripheral parts and of an intense fibrotic reaction in the deeper parts; this first stage is followed by regeneration of pulmonary tissue and a gradual absorption of the fibrous scar tissue. The second stage, marked by resolution of fibrosis, corresponds to the final stage of healing and is observed only after disappearance of the infective agent from the lesion; it is described as the seal on the cure of the bacillary process. Cavities are obliterated or diminished in size by a process of regeneration of the pulmonary parenchyma combined with cicatrization, and the secretion is dried up or diminished. In acuter lesions or diffuse infiltrations a clearing process may occur without much subsequent fibrotic reaction. In all types of disease the onset of the cicatricial and restitutive process is attended by abatement of the fever and the symptoms of intoxication, and objective improvement in the general condition is reflected in a subjective sensation of well-being. A number of persons suffering from extrapulmonary tuberculosis were also treated in the same manner as those whose disease was in the lungs, and in most cases good results were noted.

It is unfortunate that the results of the treatment are presented without any attempt at systematization (except in a report from Johannesburg Hospital), and it is difficult to arrive at a clear assessment of its value. It might be deduced from the article, too, that cases in which the response to treatment was most satisfactory have been selected for detailed description, and in these it is not clearly stated whether adjuvant treatment of a general character, such as rest in bed, was instituted at the same time as the injections of endotoxoid were commenced. The illustrative skiagrams are so poorly reproduced as to be of little assistance to the critical reader. Nevertheless there can be no doubt that the satisfactory results of treatment with tuberculosis endotoxoid in many of the patients who were very ill with advanced and progressive disease could not have been expected of any other method of treatment hitherto at our disposal. It is desirable that the clinical records of all the patients treated should be reviewed in greater detail; and it is to be anticipated that the encouraging results achieved in South Africa will stimulate investigation on a much larger scale of this new specific agent in the treatment of tuberculosis.

[1] *Tubercle*, June, 1939.

PROSTATECTOMY AT THE AGE OF 110.

J. BAYARD CLARK has reported successful prostatectomy in a patient aged one hundred and ten years.[1] Clark was at considerable pains to confirm the patient's statement about his age. Inquiries showed that he was more than 107 or 108 years old, and that his own estimate of 110 years was probably correct. The patient was a negro who was born in slavery. He was admitted to hospital suffering from acute retention of urine of four days' duration, and had had no previous difficulty or urinary disturbance. The dome of the bladder reached the umbilicus. The prostate was smooth, evenly lobulated, elastic and "about the size of a tangerine orange". Gradual decompression was instituted. The bladder was then drained suprapubically. Three weeks later prostatectomy was performed, the gland being nucleated and the prostatic bed being packed with a rubber dam. Apart from transient epididymitis, recovery was uneventful. Clark draws a "moral" from this case, that "perhaps the first hundred years of life are the worst after all".

[1] *The Journal of the American Medical Association*, August 12, 1939.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Pneumonia.

R. L. CECIL (*Bulletin of the New York Academy of Medicine*, February, 1939) discusses the present status of serum therapy in pneumonia. The author describes the development of serum therapy with Type I serum. It has, he states, reduced the fatality rate of Type I pneumonia from the standard 30% to less than 10% when only early cases are dealt with, and it has reduced the mortality by half in all cases. Felton's concentration of the serum to contain five or ten times as much antibody per unit of volume as the original serum is of great assistance. Neufeld's method of sputum testing has made it possible to ascertain the type of pneumococcus from the sputum within an hour or two, and a further advance has been the introduction of antipneumococcal rabbit serum. It is doubtful at present whether this is more valuable than horse serum. The strength of serum is measured in units per cubic centimetre, the unit being that amount of antibody which protects a white mouse against one million fatal doses of virulent homologous pneumococcus culture. This is usually dispensed in vials containing 10 to 20,000 units. Serum should be used in every case of pneumonia in which a definite pneumococcus type is determined and in which serum is available. The only contraindications are terminal pneumonia and severe allergic states. Patients with very low blood pressure may be given glucose and saline solution intravenously before a start is made with serum therapy. Children with pneumonia of Types I, VI, XIV and XIX respond well to serum given intravenously and intramuscularly. Patients should be carefully questioned as to any previous history of asthma, hay fever, urticaria or serum injections. Intradermal tests are made with one drop of a 1 in 70 dilution of serum; conjunctival tests made with one or two drops of a similar dilution placed in each eye are necessary. These should be read fifteen or more minutes later. A positive response to the conjunctival test contraindicates serum therapy, as does a positive reaction to the skin test. Desensitization is dangerous to highly sensitive patients. Slight sensitiveness may be overcome by the injection of 0·5 to 1·0 cubic centimetre of adrenaline ten minutes before serum therapy is begun; small increasing doses of well-diluted serum are then given. Urticaria may occur during administration of the serum; the administration is then suspended for a short time. If sensitivity tests elicit no response one cubic centimetre of serum diluted with nine cubic centimetres of saline solution is given very slowly into a vein, the colour and pulse rate being carefully watched. One cubic centimetre of adrenaline should be prepared in a syringe for any emergency. If after one hour there is no reaction from the small diluted dose of one cubic centimetre, 10 to 20 cubic centimetres are given and the dose is repeated until 100,000 units have been given. In bacteriæmic cases 200,000 units should be given in the first twelve hours. A failure to respond to treatment within twenty-four hours suggests an error in typing of the pneumococcus, which is fairly common. It may be necessary to give more serum if the pulse, temperature and general condition do not improve. This is often the case with patients over forty-five years of age, when more than one lobe is affected and when serum treatment is begun after the third day. The use of serum is continued until the temperature and pulse rate are normal or until the intradermal or agglutination tests elicit positive reactions. Serum therapy should not be recommenced if it has been suspended for several days, in view of the danger of anaphylaxis. The various reactions to serum therapy are well known; acute allergy, occasionally fatal, is relieved usually by the subcutaneous injection of 0·5 to 1·0 cubic centimetre of adrenaline, well rubbed in. Chills, hyperpyrexia and profuse perspiration with shock and stupor may occur. Serum sickness and urticaria, occurring in 15% to 20% of cases, are less serious. The results recorded indicate a pronounced lessening of mortality in pneumonia caused by pneumococcus Types I, II, V, VII, VIII and XIV when serum is used. "Dagenan" or "M & B 693" has recently been advocated. The author favours a combination of serum and "Dagenan". He observes that in private practice the use of serum is less frequent, because the condition of private patients is not so fully investigated bacteriologically, because serum is given later and in inadequate doses, and because there is a higher incidence of Type III pneumonia in private practice. The results in private practice are not nearly so good as those obtained in hospital practice.

High Protein Diet in Peptic Ulcer.

C. WINDWER AND M. J. MATZNER (*American Journal of Digestive Diseases and Nutrition*, January, 1939) point out the theoretical basis for the use of a high protein diet in the treatment of peptic ulcer. They state that protein is able to neutralize acids by its acid-binding properties and the inhibition of peptic activity. They also refer to their own experimental work to show that supplementary protein feedings to animals have prevented pepsin-hydrochloric acid gastric ulcers. Forty patients were selected from their clinic and given a bland, high caloric and high protein diet, consisting of 150 grammes of protein, 100 grammes of fat and 200 grammes of carbohydrate. The protein consisted of lean boiled meat, chicken, cheese, milk and gelatine. The last named was given in doses of eight grammes every hour between feedings for seven doses. About 90% of the group obtained symptomatic relief, which was maintained for a long period. This percentage is higher than is found with spontaneous remissions. All the patients gained weight with the high caloric diet. The high protein content may produce an increased flow of bile salts; the gelatine by its hydrophilic colloid action may lessen gastric irritation by absorbing the digestive secretion of the stomach. The high glycine content of gelatine may contribute to the rapid healing of the ulcer, as it is a good muscle and tissue builder.

Desoxycorticosterone.

THORN AND ENGEL EISENBERG have reported that subcutaneous insertion of two tablets of 100 milligrammes each of desoxycorticosterone acetate would maintain in good health for four or more months dogs whose adrenal glands had been removed. Apparently about one milligramme was used per day. Levy Simpson (*Proceedings of the Royal Society of Medicine*, April, 1939) has reported the successful use of implanted tablets in the treatment of four patients with Addison's disease, four tablets of 50 milligrammes of the above-named drug being used in each instance. The effect lasted for about three months. Extra effort or infection necessitated an increased supply.

Chronic Superficial Gastritis.

THE gastroscopic examination of the stomach has helped to clarify the features of gastritis; a number of writers have adopted the classification of Schindler and Ortmayer into superficial, hypertrophic and atrophic types. A study of the superficial type with regard to clinical features and gastric function has been made in a series of 50 cases by J. Bank and J. F. Renshaw (*The Journal of the American Medical Association*, January 21, 1939). The gastroscopic diagnosis was based on the observation in varying degrees of reddening of the mucosa, with patches of adherent greenish-grey to white exudate; œdema evidenced by a swollen, moist, boggy appearance; diffuse or punctate superficial hæmorrhages; erosions and evidence of friability of the mucosa. In half the cases a syndrome of duodenal ulcer was present; in the remainder the symptoms were not considered characteristic. A long history of dyspeptic symptoms and loss of weight are constant features; foci of infection were found in 86% of cases, whereas tobacco and alcohol did not play any important ætiological part in this series. According to the ordinary two-hour fractional test meal, 50% of patients in this series had hyperchlorhydria and 26% had no free acid

in the gastric juice. These findings are contrary to the opinions usually held, and the question is naturally asked whether this superficial or catarrhal stage is one in which the acid-secreting cells are stimulated, only to be depressed in the more advanced types. To determine the motility, the contents of the stomach were aspirated at the end of the two hours after the test meal and measured. In 28 cases delayed emptying was present; most of these patients had hyperacidity, only three having achlorhydria. Rapid motility with achlorhydria is well known; and it is interesting to note here the effect of the gastritis, which seems to delay the motility. This fact has been noted by other observers.

NEUROLOGY AND PSYCHIATRY.

Hemiatrophy of the Brain.

BERNARD J. ALPERS AND RICHARD B. DEAR (*The Journal of Nervous and Mental Disease*, May, 1939) record in detail a case of hemiatrophy of the brain. The patient, who was feeble-minded, died at the age of twenty-two years. During life he had manifested right hemiplegia and convulsions. They also review the recorded cases of this rare condition, and demonstrate that the term hemiatrophy has been applied to a wide variety of cerebral abnormalities produced from many different causes. The authors, for this reason, propose a classification into (i) primary or congenital groups and (ii) secondary or acquired groups. The first group should include cases in which the symptoms must have been present from birth; in the second group are placed those cases which have developed from a variety of causes in later life. The authors discriminate between hemiatrophy and lobar atrophy and the loss of brain substance from vascular accidents.

Severe Dementia Associated with Bilateral Symmetrical Degeneration of the Thalamus.

K. STERN (*Brain*, Volume LXII, Part 2, 1939) reports a case of neurological interest for which no analogy could be discovered in medical literature. The patient was a male of forty-one years, whose family history was clear and who had had no previous illness, accident or operation, and who, nine months before admission to hospital, was active and healthy. He first complained of fatigue, became more inert and commenced licking his lips and complaining of dryness of the mouth. Thirst and polyuria ensued and the drowsiness became progressively more pronounced. He spoke less and less, and took increasingly less interest in his affairs. His movements became gradually slower until finally he was quite inert. His memory was poor. He talked nonsense. All his symptoms grew rapidly worse until he presented a picture of profound dementia and the final coma supervened. Neurological examination, with the exception of loss of the pupillary reflexes and forced sucking and grasping movements, revealed no abnormality. The cerebro-spinal fluid presented no abnormalities, and a radiogram of the skull revealed nothing abnormal. The clinical picture, together with a detailed *post mortem* study of the cerebral pathological change, showed that the disease had nothing in common with cerebral syphilis, cerebral tumour, circumscribed cortical atrophy or the diffuse presenile degeneration described by Alzheimer. Microscopic examination revealed a selective bilateral symmetrical degeneration of the thalamus; but the arcuate, ventral and mid-line nuclei and the lateral and medical geniculate bodies were uninvolved. The author believes that a new and peculiar system disease has been described, the ætiology of which remains entirely undiscovered.

Use of "Metrazol" in the Functional Psychoses.

CHARLES F. READ, LOUIS STEINBERG, ERICH LIEBERT AND ISIDORE FINKELMAN (*The American Journal of Psychiatry*, January, 1939) have demonstrated the value of "Metrazol" shock treatment in other psychotic states than schizophrenia. They found, in studying the physiological action of "Metrazol", that the blood sugar level falls just prior to the convulsion and then rises sharply with the occurrence of the convulsion. A general and profound alteration in the biochemistry of the body is produced. Two and a half hours after the seizure the blood constituents return to normal. Mental efficiency remains low for some time after the fit and normal efficiency does not fully return for from four to eight hours after the administration of "Metrazol". It is suggested that other unknown factors, apart from the anoxæmia which stimulates the sympathetic nervous system, are responsible for the amelioration of the mental symptoms of patients treated by "Metrazol".

Therapeutic Significance of Fear in the "Metrazol" Treatment of Schizophrenia.

THE object of the investigation undertaken by Louis H. Cohen (*The American Journal of Psychiatry*, May, 1939) was to determine whether fear played any therapeutic part in the "Metrazol" ("Cardiazol") shock treatment of schizophrenia. The reactions of twenty patients were studied and compared. In order to induce fear the patients were given the intravenous "Metrazol" injection so slowly that no convulsion occurred but the preconvulsive "auræ" and all the unpleasant subjective symptoms would be induced. This procedure was carried out on ten successive days and the therapeutic outcome at the end of a month was compared with that which followed a similar period in which full shocks were induced. It was thereby clearly shown that better results followed the use of full shocks and that fear associated with the treatment was of minor therapeutic value.

Mental Hygiene at Senescence.

GEORGE LAWTON (*Mental Hygiene*, April, 1939) reviews the psychiatric and sociological aspects of old age and finds a general and lamentable lack of knowledge regarding the psychology of senescence. An approach to the psychological difficulties peculiar to old age may be made along several lines: (i) problems presented by neurotic, pre-psychotic, psychotic, feeble-minded and deteriorated individuals; (ii) the minor maladjustments of reasonably adequate old people caused by a variety of circumstances, the chief of which may be excessive economic pressure and inhibitory social attitudes; and (iii) the stresses and strains of those who are undergoing the normal involutional changes. Much may be done to help those who fall into the last two categories, and much of the frank mental trouble occurring in older people is theoretically preventible if rightly assessed in earlier life. Because of a disinclination to understand the problems arising with the dimming of the mental powers of the aged much needless suffering is perpetrated and senility is approached with dread. A strong plea is made for more intensive study of the abilities, recreational activities and emotional problems of the aged. Geriatrics might be usefully incorporated in the medical curriculum, so that future physicians need not so grossly neglect the troubles and sorrows of the aged. Many of the problems of senility are closely bound up with economic factors which need modification, but which cannot be dealt with until the underlying psychiatric factors have been more thoroughly assessed, investigated and assimilated. Guidance becomes as necessary to individuals at the close of life as it was to children at the beginning.

The Potassium Content of Muscle in Disease.

J. N. CUMINGS (*Brain*, Volume LXII, Part 2, 1939) presents data concerning the potassium content found in muscles in a variety of neurological disorders, and more especially in such hitherto unexplored conditions as *dystrophia myotonica* and *myasthenia gravis*. The content of potassium was found to vary, being below normal in cases of *dystrophia myotonica* and higher than normal in *myasthenia gravis*. It was also found that prostigmin produced an increase in potassium in myotonic and reduced the content in myasthenic muscles.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Melbourne Hospital on May 17, 1939. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Part of this report appeared in the issue of September 16, 1939.

Anuria due to Calculus.

DR. G. R. A. SYME showed a male patient, aged thirty-nine years. Two and a half years earlier the patient had had a vesical calculus removed suprapubically at another hospital, and shortly after the operation he had passed several other calculi *per urethram*. For one year after the operation he had had a dull ache in the left loin, with attacks of shivering and sweating.

The patient was admitted to hospital in April, 1938, suffering from pyelitis and left renal colic. Radiographic examination revealed some enlargement of the left kidney and two calculi in the left ureter, just outside the bladder. Under general anæsthesia the bladder was opened and the left ureter was dilated; two calculi were removed. The bladder was drained. The kidney diminished in size and the infection was reduced by antiseptics, but not entirely eliminated. The stone examined was found to consist of triple phosphates.

The patient reappeared at the hospital ten months later, having been in fairly good health until a short time previously, when pain in the left loin and vomiting recurred. The left kidney was large and tender. A diagnosis of left pyonephrosis was made. Radiographic examination revealed a large calculus just outside the pelvis of the left kidney and also a small calculus in the lowest calyx of the right kidney. The function of the right kidney was normal, but there was no efflux of urine from the left kidney.

Under spinal anæsthesia the left kidney was exposed. A large perinephric abscess and a pyonephritic kidney were observed. It was deemed dangerous to remove the kidney at the time, so nephrostomy was performed and drainage was instituted. The patient's condition improved satisfactorily, and Dr. Syme was on the point of removing the kidney when the patient had an attack of right renal colic and anuria for twenty-four hours. Radiographic examination showed that the stone in the right kidney had moved down and was blocking the right ureter. A ureteric catheter was passed; it failed to pass the stone or to move it, so an immediate operation was performed, again under spinal anæsthesia. The right ureter was exposed and the stone was removed. It was composed of calcium oxalate. The patient stood the operation well, and a fortnight later the left kidney and ureteric calculus were removed.

Dr. Syme said that the patient had been extremely ill and two blood transfusions were given; his hæmoglobin value was only 34%. His condition gradually improved, and although his urine still contained *Bacillus coli communis*, he was discharged a month later. Microscopic examination of a section of the kidney revealed giant cells, and the kidney was reported to be tuberculous. However, no tubercle bacilli were grown in attempted culture from the urine, and radiographic examination of the chest revealed no abnormality. Tests of the patient's calcium metabolism had not been carried out up to the time of the meeting.

Subdural Hæmatoma.

Dr. Syme's second patient was a man, aged thirty years, who, five weeks before his admission to hospital, had fallen while at work and struck the back of his head violently on a concrete floor. He had not lost consciousness, but felt drowsy and had a headache. The symptoms had persisted and the headache had gradually become more severe. For the last four days the patient had had persistent vomiting, unrelated to meals, and "spots before the eyes". He had had attacks of faintness and weakness in the limbs, falling directly forwards. His mental condition had deteriorated, and at the time of the meeting he was drowsy and confused.

On admission to hospital the patient was semi-stuporose. His temperature was 99·2° F. and his pulse and respiration rates 54 and 18 per minute respectively. His systolic blood pressure was 140 and his diastolic blood pressure 96 millimetres of mercury. His pupils were small but equal, and reacted to light and accommodation. Examination of the nervous system revealed that all tendon reflexes on the left side were slightly diminished and the superficial abdominal reflexes were equal and active; the right plantar reflex was flexor in type, while the left was equivocal. There was no change or inequality in muscle tone. A diagnostic lumbar puncture was made; the fluid was under a pressure of 220 millimetres of water; the pressure rose to 300 millimetres on jugular compression, and then rapidly returned to its previous level. Operation was decided upon, in spite of the doubt concerning the localization of the lesion.

The anæsthetic agent was ether, administered by the intratracheal route. The right parietal area was trephined; the dura was tense and not pulsating, and aspiration was without result. A trephine hole was then made over the occipital area, where the dura was normal and pulsating. Finally the left fronto-parietal area was explored and a tense non-pulsating dura was exposed and incised. A large semi-fluid clot was evacuated. The cavity was irrigated and closed without drainage. After the operation the patient's condition progressed rapidly, and five days later he appeared quite normal.

Hydatid Cyst of the Liver.

Dr. Syme then showed a female patient, aged twenty-five years, who had been admitted to hospital with a history of pain in the right costal margin and right lower portion of the chest, of three weeks' duration. She had a slight cough but no sputum. A fortnight later jaundice developed and the stools became clay-coloured.

The patient's past history was interesting. At the age of three years a hydatid cyst had been removed from her liver at the Children's Hospital.

Examination revealed considerable enlargement and tenderness of the liver and slight dulness at the base of the right lung. Both the Casoni test and the complement fixation test elicited strongly positive reactions. Radiographic examination revealed two distinct cavities below the diaphragm in the substance of the liver.

At operation the old abdominal scar was reincised and the liver was palpated; a cyst was felt under the costal margin. The peritoneum was sewn to the liver and the cyst was then incised extraperitoneally; pus, daughter cysts and membrane were evacuated and a large drainage tube was inserted. Drainage was satisfactory and the patient's temperature fell to normal. Two weeks later another radiographic examination was carried out for visualization of the site of the other cyst. The skiagram showed a cavity with numerous daughter cysts. The movement of the patient to the X ray room apparently caused this second cyst to rupture into the cavity left by the first cyst; next morning a complete cyst wall and daughter cysts were found on the dressings. The cavity was enlarged and further débris was removed. The drainage tube was reinserted. At the time of the meeting slight drainage was still taking place, but the patient was very well.

Lung Abscess.

Dr. Syme's next patient was a male, aged thirty years, who had been admitted to hospital on June 14, 1938, with a history of pain in the back and left side, worse on breathing, hæmoptysis, and cough with foul purulent sputum, of eight weeks' duration. Prior to the onset of pain he had been perfectly well, but he had had pneumonia and pleurisy twelve years previously.

No tubercle bacilli were found in the sputum. Radiographic examination revealed no cavity; but a large opaque area was seen in the lower lobe of the left lung, into which lipiodol did not penetrate. There was no reaction to the Wassermann test.

The patient had repeated hæmoptyses during the next four months, but the amount of sputum gradually decreased with postural drainage. The area of dulness seen radiographically did not decrease in size, but a fluid level became visible. The patient was discharged on August 17, 1938.

He was readmitted to hospital on October 31, 1938. He had been very well until two weeks previously, although he had still had a cough with a small amount of sputum; at that time, however, he became dyspnœic on exertion and had profuse night sweats, pain being again present in the left side.

On examination the patient was found to have a temperature of 99° F., and signs suggesting consolidation at the base of the left lung were present. Radiographic examination revealed that the area of dulness at the base of the left lung had slightly increased in size; this was regarded as pneumonitis surrounding an abscess. The patient's condition improved slowly and his temperature fell to normal. A bronchoscopic examination was made on November 12, 1938; much foul pus drained away, and acute inflammation of the mucosa of the lower bronchi was seen. The patient continued to cough up purulent sputum; his temperature again rose and his general condition deteriorated, in spite of repeated bronchoscopic drainage. The amount of sputum remained at about four or five ounces daily.

On January 17, 1939, operation was performed under spinal and nitrous oxide anæsthesia. Part of the seventh rib was resected and an abscess cavity about two inches in diameter was opened and drained; continuous negative pressure drainage to the pleural cavity was instituted. There was very little drainage from the pleura, but free drainage from the abscess cavity took place over the next two weeks. During this period the patient's temperature gradually fell to normal and his general condition improved. He had some neuritic pain in the back and shoulder, probably due to involvement of the intercostal nerves in scar tissue. At the time of his discharge on March 17, 1939, he had no cough or sputum, had gained in weight and looked well. Radiographic examination revealed an area of dense fibrosis in the lower lobe of the left lung.

Paralytic Ileus.

Dr. Syme next showed a female patient who had undergone total hysterectomy for chronic pelvic infection. The operation had been followed by excessive vomiting, but the bowels were open and no abdominal distension was present. However, by the fifth post-operative day distension was excessive and vomiting copious; the vomitus was foul and definite ileus was present. The patient was treated by continuous gastric suction by means of Rehfuss's tube, and the intravenous administration of glucose and saline solution. The treatment was persisted in for fourteen days, but the patient's symptoms were not relieved. By this time she was *in extremis*; she was vomiting fæcal matter, was cyanosed, and her pulse was thready, the rate being 140 per minute. At this stage the Miller-Abbot double lumen tube was inserted and passed into the pylorus in eight hours and then on down the small bowel until it was seen radiographically to be seven feet down the intestine. Eight and a half pints of foul fæcal fluid were sucked off and the patient improved dramatically. After forty-eight hours she felt so well that she pulled the tube out herself. This was followed by a return of the vomiting and abdominal distension. The Miller-Abbot tube was reinserted and four pints of foul fluid were removed. Then the return fluid became clear, the patient's bowels opened and the tube was removed. She made an uninterrupted recovery. The total output of fluids was 49 pints; the total intake by mouth and rectally and intravenously during the same period was 47 pints.

Carcinoma of the Scrotum.

Dr. Syme finally showed a male patient, aged fifty-five years, who had been working for fifteen years at tar refining and frequently spilt tar on his trousers. At the time of the meeting he presented a typical carcinoma of the scrotum. No inguinal glands were palpable. Dr. Syme said that he proposed to resect the scrotal tissue only, because of the low-grade malignancy of the condition and the risk of œdema of the legs if the glands were resected.

Physical Therapy.

DR. L. T. WEDLICK showed several patients to illustrate some results of physical therapy.

The first patient was a male, aged fifty-five years, who had suffered from intermittent claudication and for two months had had painful cramp in the calves, which prevented him from walking more than 30 to 50 yards. Short-wave therapy was given daily to the feet, and after two weeks he was able to walk 300 yards. Treatment was given for a further three weeks, and the patient was then able to walk 400 yards and was capable of working. He was discharged. When seen three months later he was still working and had had no relapse.

Dr. Wedlick's second patient was a man who had suffered from a chronic infection of the finger after trauma. For three months surgical treatment was carried out; the nail was removed and dressings were applied. Treatment with ultra-violet light was then given locally on alternate days and the finger healed in two weeks.

The next patient shown by Dr. Wedlick was a female, aged forty-seven years, who for years had suffered from asthma and had tried all the usual remedial measures. Short-wave treatment was given on alternate days for three months. At the time of the meeting the patient was using only one-third of the amount of adrenaline that she had used at the commencement of treatment.

Dr. Wedlick then showed a female patient, aged forty-five years, who had suffered from asthma for twenty years and whose condition six years previously had been looked upon as hopeless after all the usual measures had been tried. Short-wave treatment was applied through the chest on alternate days for ten months. The patient was relieved from the commencement of treatment and had no further attacks during treatment. The treatment had been concluded six months prior to the meeting and the patient was still free from attacks.

Dr. Wedlick's next patient was a female, aged thirty years, who had suffered from hay fever. Two years previously she had been treated with the Kromayer quartz applicator. Treatment had been directed to the nose and had been carried out twice a week for six weeks; the patient had experienced considerable relief. She had reported again six months prior to the meeting, and the course of treatment had been repeated with exactly similar results.

The next patient shown by Dr. Wedlick was a female, aged forty years, who had had a traumatic ulcer, three inches by six inches in area, on the calf of her left leg. She had been treated in the out-patient department with stockings and dressings for one month; the condition of the ulcer was unchanged at the end of that time. Ultra-violet light was then applied locally on alternate days and the ulcer healed in sixteen days.

Dr. Wedlick then showed a female patient who had had left antral sinusitis for years. Three years previously she had undergone an antrostomy operation and had had wash-outs since. Radiographical examination on April 29, 1938, had revealed considerable thickening of the mucosa in the left antrum. Short-wave treatment was then given on alternate days for one month. A further radiographic examination on May 31, 1938, revealed that the antral thickening had greatly diminished. The patient's clinical improvement was correspondingly great, and was sufficient to enable her to be discharged. She had had no relapse since.

Dr. Wedlick finally showed a male patient who had had chronic sinusitis for a number of years. Radiographic examination on December 9, 1937, had revealed thickening of the mucosa in the right antrum. He was treated in the

ear, nose and throat departments by means of wash-outs *et cetera*, and on March 15, 1938, the condition of the antrum was unchanged. He was given short-wave treatment on alternate days for one month. Radiographic examination on April 12, 1938, revealed considerable improvement in his condition. The patient's clinical improvement was correspondingly great; he was discharged and had not needed treatment since.

A Fever Cabinet.

Dr. Wedlick also showed a fever cabinet which had been recently completed to enable systematic fever investigation to be carried out in suitable cases of gonorrhœa, general paralysis of the insane, chronic rheumatism *et cetera*. The cabinet was a modified Kettering cabinet, and consisted of a heat insulated box through which was circulated hot moist air, driven up from below by a blower. The air temperature inside the cabinet was controlled automatically by a thermostat; this regulated the heating element and enabled the air temperature to be read off and to be preset at any desired figure. The humidity could be read off by the use of a wet and dry bulb thermometer registering outside the cabinet. The patient's temperature was shown by a continuously recording electrical rectal thermometer, which was read outside the cabinet on a drum; this obviated any necessity for interfering with the patient, and enabled the temperature to be seen at a glance at any time throughout the treatment. The patient could be watched through an observation window at the head of the cabinet, provided with a windscreen wiper. Provision was made for instant access to the patient if desired, and his head could be cooled by an electric fan. Dr. Wedlick pointed out that with this cabinet it would be possible to maintain the patient's temperature at any desired point, as high as 106° or 107° F., for any given period up to ten hours or longer.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 22, 1939, at the Royal North Shore Hospital of Sydney. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital.

Congenital Purpura.

DR. W. WILSON INGRAM showed a female patient, aged forty-eight years, who had been admitted to hospital on May 6, 1939, suffering from painful hæmorrhages into both legs.

The patient belonged to a family in which all the females were affected and in which the males were normal. The patient was first admitted to hospital nine years ago for uterine hæmorrhage. Previously she had undergone curettage of the uterus on four occasions. Owing to profuse hæmorrhage subtotal hysterectomy was performed, the cervix being left. Severe secondary hæmorrhage occurred ten days after operation. After this the patient had numerous hæmorrhages into the legs at intervals of a few months. Blood counts made at this time showed that the erythrocytes numbered over 4,500,000 per cubic millimetre and the number went as high as 8,000,000. The spleen was exposed to irradiation by X rays on two occasions without relief of symptoms. In 1927 the patient was treated for hæmorrhage by blood transfusion and relief followed for some months, the blood count revealing 4,000,000 erythrocytes per cubic millimetre. The patient had had repeated hæmorrhages since that treatment by transfusion. The patient's mother died at the age of thirty-five years from cerebral hæmorrhage; her sister died at the age of twenty-five years from hæmorrhage after child-birth; a sister suffered from frequent hæmorrhages after cuts *et cetera*, and almost died from hæmorrhage at child-birth. The patient's daughter was a "bleeder".

At the time of her admission to hospital the patient had been free from hæmorrhages for the past eight years; she then had an acute shivering attack with pain in the epigastrium followed by vomiting of bile. She had been vomiting bile at intervals for the last three years, and stated that she could not tolerate fats or carbohydrates. She had been unusually tired for several days before her admission to hospital. Five days after her admission she had subcutaneous hæmorrhages into the right leg. The leg was acutely painful. She perspired freely and was gravely exhausted. Four days later she had extensive hæmorrhages in both legs and complained of great pain.

The patient had red hair and was pale. Her tongue was raw, of the "beef steak" type. The systolic blood pressure was 150 and the diastolic pressure 80 millimetres of mercury. No abnormality was detected in the heart. There was a generalized tenderness over the whole abdomen, and the spleen was enlarged and tender. The erythrocytes had numbered 5,700,000 per cubic millimetre at the time of her admission to hospital; three weeks later they numbered 5,400,000, and at the time of the meeting they numbered 4,600,000 per cubic millimetre. The bleeding time was two minutes and the blood coagulation time nine minutes. Platelets numbered 233,000 per cubic millimetre. The patient had been given sedatives, hæmostatic serum and calcium with vitamin *D*. She had received a blood transfusion one month prior to the meeting; since then there had been no fresh hæmorrhages.

Malignant Arterial Hypertension.

Dr. Ingram next showed a female patient, aged twenty-nine years, who had been admitted to hospital on May 10, 1939. She had had acute anterior poliomyelitis at the age of ten months. Nine years before her present admission to hospital, at the birth of her second child, she had had œdema of the face and legs and had lost the power of her right arm for one week. She had four children, aged ten, nine, six and five years respectively; all were healthy. She had been subjected to hysterectomy three years prior to the meeting.

The patient stated that five years previously she had begun to suffer from headache and became easily tired and breathless on exertion. At times the headache was very severe and was accompanied by nausea and vomiting. After standing all day she noticed swelling of her legs. The symptoms gradually became more pronounced until a few days before her admission to hospital, when she developed severe occipital headache and pain in the neck. She also had severe pain behind the eyes, accompanied by photophobia. Cough and sputum had been present for one week; and she had pain in the left side of the chest, which was accentuated by deep breathing.

On admission to hospital the patient was extremely drowsy and could be roused only with difficulty. She complained of vertigo, nausea and severe headache. She refused all food, and any attempt to sit up brought on extreme nausea.

A physical examination was carried out. The arteries were found to be thickened and tortuous; the systolic blood pressure was 250 and the diastolic pressure 170 millimetres of mercury. The heart was enlarged in the sixth intercostal space in the anterior axillary line. The second aortic sound was accentuated. There was no œdema. An electrocardiographic examination revealed no abnormality. The specific gravity of the urine was 1010; the urine contained no albumin and no abnormal constituents. The blood urea content was 30 milligrammes *per centum*. The blood creatinine level was 0·9%. The optic fundi were normal. No abnormality was detected in the chest or abdomen.

Wasting of the upper part of the right arm and of the left foot was present, as a result of the poliomyelitis; but apart from that no abnormality was detected in the central nervous system. A full blood count revealed no abnormality. The cerebro-spinal fluid was found to be under pressure, but contained no abnormal constituents. Twenty cubic centimetres of cerebro-spinal fluid were removed; this procedure relieved the patient's headache. She was given intravenous injections of "Calcibronat", and a sedative mixture was exhibited. At the time of the meeting the patient was free from symptoms. On May 10, 1939, the systolic blood pressure was 250 and the diastolic pressure 170 millimetres of mercury; these figures gradually fell, until eventually, on June 17, 1939, the systolic blood pressure was 160 and the diastolic pressure 110 millimetres of mercury.

Acute Hæmolytic Crisis.

DR. B. T. SHALLARD, in conjunction with DR. R. A. MONEY, showed a man, aged forty-three years, suffering from acholuric jaundice, who had had an acute hæmolytic crisis treated by splenectomy. Shortness of breath had been present for three weeks, and there were giddiness, easy bruising and some swelling of the ankles. There was no family history of anæmia. The patient was very pallid and slightly jaundiced, and on his admission to hospital the liver, spleen and lymph glands were not palpable. A blood count gave the following information: the erythrocytes numbered 1,300,000 and the leucocytes 11,000 per cubic millimetre; 62% of the erythrocytes were reticulocytes; platelets numbered 437,000 per cubic millimetre and the hæmoglobin value was 28%. The bleeding time was one and a half minutes and the blood coagulation time three minutes. A test of the fragility of the red cells showed that hæmolysis occurred in 0·75% to 0·3% saline solution. A macrocytic anæmia with numerous red cells (spherocytes) was present. The plasma bilirubin content was two milligrammes *per centum*, and the urine did not react to the test for bilirubin.

The spleen was first palpable eight days after the patient's admission to hospital. Blood transfusions were given. As the condition of the patient became more grave, splenectomy was performed and 2,100 cubic centimetres of blood were given by transfusion on the ninth day after his admission to hospital. The patient improved immediately, as was shown by a blood count made two days later; at that time the erythrocytes numbered 3,500,000 and the leucocytes 11,300 per cubic millimetre; 14% of the erythrocytes were reticulocytes, and the erythrocytes were less irregular in size. Spherocytes were fewer; and hæmolysis of erythrocytes occurred in 0·65% to 0·3% saline solution. Four months later, at the time of the meeting, the patient's blood picture was being fairly well maintained. The erythrocytes numbered 3,000,000 and the leucocytes 7,800 per cubic millimetre; the hæmoglobin value was 56%, and 14% of the erythrocytes were reticulocytes. Hæmolysis occurred in 0·6% to 0·3% saline solution. The plasma bilirubin content was 2·5 milligrammes *per centum*.

Aplastic Anæmia.

Dr. Shallard's second patient was a woman, aged fifty years, suffering from aplastic anæmia. She had been admitted to hospital complaining of weakness and lassitude, dyspnœa on exertion, palpitation, tinnitus, petechial hæmorrhages, epistaxis and melena on one occasion. The skin and mucous membranes were pale and the sacrum and ankles were œdematous. The liver, spleen and lymph glands were not palpable. A blood count revealed profound anæmia; the erythrocytes numbered 1,000,000 and the leucocytes 6,500 per cubic millimetre; the hæmoglobin value was 25% and platelets numbered 52,000 per cubic millimetre. There was variation in the size and shape of the erythrocytes, but normoblasts and megaloblasts were completely absent, and reticulocytes were present only in negligible amounts; they never numbered more than 1·5% of the erythrocytes in repeated blood counts. The bleeding time was more than fifty minutes and the blood coagulation time was eight minutes. A test of the fragility of the erythrocytes elicited normal results, and a fractional test meal revealed a normal acid curve. The result of the capillary resistance test was positive. Intensive treatment with concentrated liver extract and iron caused no improvement in the blood picture, which could be maintained only by transfusions. Bone marrow taken for examination by sternal puncture was found to be orthoplastic.

Gastrostaxis.

Dr. Shallard, in conjunction with Dr. Jamieson, next showed a young man, aged nineteen years, who had had gastrostaxis which had been treated by partial gastrectomy. He had been admitted to hospital on March 25, 1939, with a history of epigastric pain on the previous day, of thirty minutes' duration. Nausea, sweating and hæmatemesis were present on the day of his admission to hospital. He had lost two pints of blood. Sixteen months previously he had had a hæmatemesis not preceded by pain. The diet had not been deficient in vitamins, and there was no family history of abnormal bleeding. No abdominal tenderness was present and the liver and spleen were not palpable. Examination by means of a fractional test meal revealed a normal acid curve; there were no other abnormal findings. The bleeding time was one and a half minutes, and the blood coagulation time was seven and a half minutes.

A blood count gave the following information: the erythrocytes numbered 4,300,000 and the leucocytes 10,600 per cubic millimetre, and the hæmoglobin value was 83%. Routine medical treatment for hæmatemesis was instituted, but the patient had further hæmatemeses on March 31, April 9 and April 18. Blood transfusions were given on April 19, after exploration, partial gastrectomy and gastroenterostomy were performed. In spite of these measures melena was present for two weeks after operation. "Clauden", "Coagulen", fibrinogen and ascorbic acid were given parenterally and the patient's condition improved. On June 1 the benzidine test for occult blood elicited no reaction.

Macroscopic examination of the portion of the stomach excised revealed no ulcerated area; but the whole mucous membrane was congested and hæmorrhagic in appearance. Sections of several areas were made. Microscopic examination of the portion of the stomach revealed vascularity of the mucosa and submucosa. Thin-walled dilated vessels were numerous, and in the mucosa extravasated blood was present in some places. There was an inflammatory reaction with plasma cells in the mucosa; this was, however, quite unbroken.

Pink Disease.

Dr. Shallard then showed a male infant, aged twelve months, suffering from pink disease. The child exhibited blepharitis and photophobia, irritability, loss of weight and loss of appetite. Sweating was present, especially about the head. The child's extremities were cold and pink. Pronounced hypotonia was present.

Osgood-Schlatter's Disease.

Dr. Shallard finally showed a boy suffering from bilateral Osgood-Schlatter's disease. He had prominent, tender tibial tuberosities. Radiological examination revealed epiphysitis with early fragmentation.

Lobar Pneumonia, Empyema Thoracis and Suppurative Pericarditis.

DR. C. W. SINCLAIR and DR. H. H. JAMIESON showed a male patient, aged thirty-three years, a boilermaker, who had been admitted to hospital on December 3, 1938. The onset of his illness had been sudden; he had had a sharp pain in the back, made worse by breathing. This had been followed in a day or two by a cough with blood-stained sputum. He was admitted to hospital on the third day of his illness. At that time lobar pneumonia was present at the base of the left lung, and the patient was very ill. An X ray examination showed that the heart was displaced to the right and that there was consolidation at the base of the left lung. The displacement of the heart was evidently a congenital condition.

On December 17, 1938, the patient was still very ill. The area of cardiac dulness had enlarged, and an X ray examination suggested pericardial effusion. Evidence of empyema on the left side gradually developed, and Dr. Jamieson saw the patient with a view to opening both pleural and pericardial cavities. On December 24, 1938, thick creamy pus was obtained by aspiration of the left pleural cavity. On December 27, 1938, the aspiration was repeated. On January 1, 1939, part of the eighth left rib was resected and the pleural cavity was drained. On January 6, 1939, resection of the fifth costal cartilage was performed and the pericardial cavity was drained. For some days the patient was desperately ill, but he gradually recovered and was discharged from hospital on March 1,

1939. At the time of the meeting he was back at work. Pneumococci were obtained from the sputum and in pure culture from the pleural and pericardial sacs.

Complete Heart Block.

DR. C. W. SINCLAIR showed a male patient, aged nineteen years, who had been admitted to hospital on July 15, 1938. He had previously had Bright's disease, at the age of three years, and pneumonia. There was also an indefinite history of three epileptic fits and of growing pains in infancy.

The patient was admitted to hospital with an eight weeks' history of breathlessness on the slightest exertion and of swelling of the abdomen, associated with dull pain in the epigastrium. He was nursed in a flat position and at complete rest. He had frequent attacks of retrosternal pain, radiating to the left and down the left arm, along with dyspnœa relieved by morphine and atropine. Associated with these attacks was severe headache. Dr. Sinclair said that on three occasions the patient had had what appeared to be a form of Stokes-Adams attack: severe retrosternal pain was followed by loss of consciousness for from half to one hour. The patient was often cyanosed, and when consciousness was regained he complained of severe headache. The heart rate varied between 36 and 40 beats per minute. The last attack had occurred at the end of March. After this attack his condition improved and the patient was able to leave his bed and walk about slowly without undue distress. During his illness the heart rate had not varied from between 36 and 40 beats per minute.

A physical examination made on his admission to hospital revealed that the pulse rate was 48 per minute; the pulse was regular in time and amplitude. Examination of the heart revealed that the precordium was flattened on the left side. By percussion it was determined that the upper border of the heart was at the level of the third rib, the right border was at the sternum and the left border was four inches from the middle line. The apex beat was four inches from the middle line in the fifth left intercostal space. Auscultation revealed a systolic murmur in the mitral area, with reduplication of the second sound; a systolic murmur was also audible in the aortic area. The systolic blood pressure was 130 and the diastolic pressure 65 millimetres of mercury. The liver was palpable three fingers' breadth below the costal margin. No abnormality was detected in the chest or central nervous system.

Another physical examination was made on June 9, 1939. The chest appeared protuberant to the left side. The pulse rate was 40 per minute; the pulse was regular and of fair amplitude. Percussion revealed a pronounced enlargement of the area of cardiac dulness. The apex beat was visible and palpable in the sixth left intercostal space, five inches from the mid-line. The liver was palpable two fingers' breadth below the costal margin. An electrocardiographic examination revealed complete heart block with myocardial degeneration. Radiological examination revealed pronounced enlargement of the heart shadow. There was no reaction to the Wassermann test.

(To be continued.)

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the University of Adelaide on July 7, 1939.

The Action of Drugs.

PROFESSOR SIR STANTON HICKS spoke on the action of drugs. In discussing pharmacology from the quantitative aspect he laid stress on the importance of simplification of the living system studied. He said that the question arose as to whether ordinary physico-chemical laws applied. Despite the vast dilution shown to be effective, there was still a large molecular population. In other words, the statistical basis for the operation of the law of mass action and the second law of thermodynamics still existed, except in some cases in which the protein molecules were concentrated in the surface and only three or four hydrogen ions were present in the cell.

The relative size of cell and effective molecule in the case of phenol and coccus was of the order of one milligramme to one ton. Even if the whole surface of a coccus was covered by phenol molecules (monomolecular layer) they would approximate only 1% of the volume of the organism.

Sir Stanton Hicks cited methods of studying cell drug reactions, the biological determination of the quantity remaining in solution after an effect had been produced being taken as the most useful. Owing to effect being mainly a surface one, micro-injection was not useful.

Minimum effective concentrations were quoted, and although low for such substances as acetylcholine and adrenaline—of the order of 1 part in 10^{11}—yet certain special sense effects were of the same order; for example, detection of fluorescein by vision. Moreover, inorganic systems showed similar astonishing effectiveness. Sir Stanton Hicks described errors in technique giving apparently high results.

He speculated on cell structure and attempted to picture the fixation of drugs. He cited Quastel's active patch views and analysed the fixation of effective doses of digitalis on cardiac muscle cells. He showed surface effect to be highly important.

Sir Stanton Hicks then criticized the current hypothesis of drug cell action; he took Traube's potential theory, the Arndt-Schulz law and the Weber-Fechner law as special examples. He entered a plea for the realization that an accuracy of more than $\pm 5\%$ could not be expected and that, therefore, almost any standard formula could be applied to results, with the production of an altogether spurious appearance of accuracy. He concluded by paying a tribute to the work of Trevan and of Clark in this field of quantitative biology.

Correspondence.

DAWES, OF THE "PATH": AN APPEAL.

SIR: Some of your readers will remember Emil Dawes, laboratory assistant in the pathological department at the Royal Prince Alfred Hospital for many years, in fact since about 1909. He was there as a lad of seventeen years when I first went down to the "Path". Of a simple, ingenuous nature, but full of energy and of a most obliging disposition, many past residents and honoraries must remember him, and many will acknowledge his readiness to help at all times when they dropped in at the obscure building with the sanguine inscription *"In cælo quies"*. He went to war with the original Fourth Battalion, and did a wonderful job on Gallipoli as medical corporal, and after that as sergeant with the Third General Hospital in Egypt and in France; in this unit he worked under Lieutenant-Colonel C. J. (later Sir Charles) Martin, one-time lecturer in physiology in Sydney and later director of the Lister Institute in London. One of his war stories was that one day "C.J." said to him: "Dawes, you need a change, take a bicycle and go out for a ride, and by the way, tie this bottle to the wheel." "C.J." wanted to try out a bike as a shaking machine for an obstinate bacterial emulsion! Some amusing stories could be told centred round Dawes, with his single-minded nature.

Well, Dawes is gone, at forty-seven years, and his widow and children not well provided for. Only a few know the good work he did all his prematurely-ending life, often working late, for the Royal Prince Alfred Hospital, for its patients and its medical staff. At his funeral some of his

old friends and associates decided to open a subscription to assist his widow and children. Subscriptions, however small, will be gratefully received and acknowledged by Dr. Edgar Thomson, Royal Prince Alfred Hospital, or by me.

Yours, etc.,
A. H. TEBBUTT.

143, Macquarie Street,
Sydney,
September 12, 1939.

M.D. DEGREES IN AUSTRALIA.

SIR: Why is it that there is such a great disparity in the value of the M.D. degree at the Australian universities? I know of many cases where a man has obtained his M.D. one and two years after graduating at the University of Melbourne, after having spent only a few months as a house physician. At Sydney and Adelaide this degree is only given to graduates of experience and culture, usually after submission of a thesis, many years after graduation. The present position is not fair, in that the M.D. degree may count considerably in the election of a candidate for an appointment.

Yours, etc.,
August 27, 1939. "WARUM."

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 76, of September 14, 1939.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH.

Temporary Appointments.—The following officers of the Emergency List are appointed for Temporary Service: *Surgeon Commander*—William Edgar Roberts, date of appointment, 31st August, 1939. *Surgeon Lieutenant*—Brian Andrew Serjeant, date of appointment, 1st September, 1939.

AUSTRALIAN MILITARY FORCES.

First Military District.

Australian Army Medical Corps.

To be Captains (provisionally) supernumerary to establishment pending absorption—Geoffrey Stanhope Hayes and Hew Fancourt Graham McDonald, 9th August, 1939, and 18th August, 1939, respectively.

Australian Army Medical Corps Reserve.

To be Captains—Edward Mansfield and William Leonard Millett, 18th August, 1939. *To be Honorary Lieutenants*—Robert Bruce Nash, Frederick Hugh Sherston Roberts, Cyril Ivett Lawrence and John Neville Coffey, 15th August, 1939.

Second Military District.

Australian Army Medical Corps Reserve.

Captain F. B. Craig is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform.

Third Military District.

Australian Army Medical Corps.

To be Captain (provisionally) supernumerary to establishment pending absorption—George Colin McKechnie, 4th August, 1939. Honorary Captain J. J. Searby is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 8th August, 1939. Captain G. J. B. Baldwin is brought on the authorized establishment, 24th August, 1939. The provisional appointment of Captain F. R. T. Stevens is terminated.

Australian Army Medical Corps Reserve.

To be Captain—Robert Milne Wishart, 15th August, 1939. *To be Honorary Captain*—George Ronald Davidson, 11th August, 1939. *To be Honorary Lieutenants*—Sydney Richardson Birch and James Neil Rattray, 23rd June, 1939, and 27th July, 1939, respectively.

Fourth Military District.

Australian Army Medical Corps.

To be Captain (provisionally) supernumerary to establishment pending absorption—Christopher Bagot Sangster, 15th August, 1939. Honorary Captain N. J. Bonnin is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally) supernumerary to establishment pending absorption, 17th August, 1939.

Fifth Military District.

Australian Army Medical Corps.

Honorary Captain D. R. C. Wilson is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally) supernumerary to establishment pending absorption, 16th August, 1939.

Australian Army Medical Corps Reserve.

The resignation of Honorary Captain W. S. Cook of his commission is accepted.

Sixth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Lachlan Neil Gollan, 18th August, 1939.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Studley Woolcott Lush, M.B., B.S., is granted a short service commission on probation with the rank of Flight Lieutenant, 11th September, 1939.

Citizen Air Force (Active Force).

Reserve.

Pilot Officer R. R. MacDonald, M.B., B.S., is transferred from the Reserve to the Active Force and from the General Duties Branch to the Medical Branch and is promoted to Flight Lieutenant, 1st September, 1939.

Medical Branch.

Flight Lieutenant R. M. Buntine, M.B., B.S., is transferred from the Reserve to the Active Force and Flight Lieutenant J. G. Brown, M.B., B.S., is transferred from the Active Force to the Reserve, 1st September, 1939.—(Ex. Min. No. 279—Approved 13th September, 1939.)

Obituary.

ARTHUR FREDERICK DAVENPORT.

WE regret to announce the death of Dr. Arthur Frederick Davenport, which occurred on September 11, 1939, at St. Kilda, Victoria.

FREDERICK JOHN GAWNE.

WE regret to announce the death of Dr. Frederick John Gawne, which occurred recently at Jeparit, Victoria.

Notice.

ADVICE has been received from the Honorary Secretary, the Institution of Engineers, Australia, Melbourne Division, that the proposed series of lectures on "Electronics", the programme of which appeared in the issue of September 2, 1939, has been abandoned.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Cummine, Harold George, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Bilton, John Herbert, M.B., B.S., 1939 (Univ. Sydney), 413, Brunswick Street, The Valley, Brisbane, Queensland.

Cox, Charles Bertram, M.B., 1937 (Univ. Sydney), Department of Pathology, the University of Sydney, Sydney.

Downes, Gwenyth Mary, M.B., B.S., 1937 (Univ. Sydney), Hornsby and District Hospital, Hornsby.

Edwards, Lando Lucas, M.B., B.S., 1937 (Univ. Sydney), 530, New Canterbury Road, Dulwich Hill.

Lockley, Ronald Paull, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.

Lyttle, John Purves, M.B., 1939 (Univ. Sydney), 276, Bourke Street, Goulburn.

Marsh, Harold Geoffrey, M.B., B.S., 1937 (Univ. Sydney), 14, Boambillee Avenue, Vaucluse.

Puddicombe, Geoffrey Henry, M.B., B.S., 1938 (Univ. Sydney), c.o. Dr. R. G. Weaver, Junee.

Reilly, Charles Patrick Cummerford, M.B., 1939 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

McGregor, Ross Alexander James, M.B., B.S., 1936 (Univ. Sydney), Ardlethan.

Mutton, John Vernon, M.B., B.S., 1933 (Univ. Sydney), 235, Macquarie Street, Sydney.

Books Received.

RECENT ADVANCES IN MEDICAL SCIENCE: A STUDY OF THEIR SOCIAL AND ECONOMIC IMPLICATIONS (THE REDE LECTURE, DELIVERED BEFORE THE UNIVERSITY OF CAMBRIDGE ON APRIL 28, 1939), by E. Mellanby, K.C.B., M.D., F.R.C.P., F.R.S., K.H.P.; 1939. Cambridge: The University Press; Melbourne: G. Jaboor. Crown 8vo, pp. 62. Price: 2s. 6d. net.

Diary for the Month.

SEPT. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 27.—Victorian Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Branch.
SEPT. 28.—South Australian Branch, B.M.A.: Branch.
SEPT. 29.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xii.

GOVERNMENT OF NORTH BORNEO: Medical Officer.
LIDCOMBE STATE HOSPITAL AND HOME, LIDCOMBE, NEW SOUTH WALES: Honorary Ear, Nose and Throat Surgeon.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Medical Staff, Clinical Assistants.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Honorary Surgeon.
STATE PUBLIC SERVICE, QUEENSLAND: Medical Officer.
THE UNIVERSITY OF ADELAIDE, SOUTH AUSTRALIA: Director in Obstetrics.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Superintendent, Resident Surgeons.
VICTORIAN RAILWAYS: Assistant Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

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